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# THE MEDICAL JOURNAL OF AUSTRALIA

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## THE EXCRETION OF LEAD AND BLOOD CHANGES IN WORKERS EXPOSED TO LEAD.

*The Division of Industrial Hygiene, Commonwealth Department of Health.*

### INTRODUCTION.

THE Australian Commonwealth Engineering Standards Association in 1926 requested the Commonwealth Department of Health to undertake certain research work regarding precautions to be taken in the use of paints containing lead. The Department, after careful consideration, decided that any investigation conducted should include the effects of exposure to lead not only on painters but on all workers handling lead in any form.

A Committee consisting of Professor H. G. Chapman, Professor of Physiology, University of Sydney, Dr. S. A. Smith, Lecturer in Medicine, University of Sydney, and Dr. D. G. Robertson, Director, Division of Industrial Hygiene, Commonwealth Department of Health, was appointed to formulate the exact lines on which the research should be conducted. The Committee agreed that as a preliminary to more complete research at a later stage the investigation should be confined to workers at the Broken Hill Associated Smelters, Port Pirie, South Australia, and to employees on the Broken Hill mines. Three lines of inquiry were adopted, namely:

1. The estimation of lead in the tissues of cadavers, this to be correlated with the industrial histories of the subjects.

2. The determination of lead in the urine of persons entering industry for the first time, and of persons who had worked varying periods in atmospheres containing lead dust.

3. The blood examination of persons entering industry for the first time and of persons who had worked varying intervals of time in industries involving exposure to lead.

The Broken Hill underground mine employees were considered particularly suitable subjects for research as an approximate estimation of the amount of lead to which they had been exposed, was possible.

The blood examinations were conducted at the Commonwealth Health Laboratory, Port Pirie, by Dr. R. Y. Mathew, Medical Officer in Charge. Urine analyses were made partly at the Commonwealth Health Laboratory, Port Pirie, by Mr. R. T. Simmons, Technical Assistant, and partly by Mr. R. W. Tannahill, B.Sc., A.A.C.I., working under Professor Chapman's directions at the Physiology Department, University of Sydney. Mr. Tannahill also performed all analyses of fæces, *post mortem* material *et cetera*. The collection of material at Broken Hill was undertaken by Dr. W. E. George, Medical Officer in Charge. Industrial histories of all examinees were recorded by Dr. George and Dr. Mathew according to the location of the examinees.

## A CRITICAL SURVEY OF THE METHODS FOR THE DETERMINATION OF LEAD IN BIOLOGICAL MATERIAL.

By R. W. TANNAHILL, B.Sc., A.A.C.I.,
*Sydney.*

### The Estimation of Lead in Urine.

In order to determine quantitatively the excretion of lead of men employed in the mining industry at Broken Hill and Port Pirie, it was necessary to find a method for the estimation of lead in urine. It was essential that the method should be capable of accurately estimating the very small amounts of lead (a few hundredths of a milligramme) which are present in the urine of persons exposed to lead.

It was found that the available methods could be placed into three groups, according to the means adopted of isolating the lead from the urine:

1. Pure chemical methods,
2. Physico-chemical methods,
3. Electro-chemical methods.

After the removal of the lead the subsequent stages for its estimation in any method of the above groups are carried out by means of various characteristic chemical reactions of lead. The means of estimation must necessarily be colorimetric, as gravimetric and volumetric methods are not capable of estimating such small amounts with any degree of accuracy.

Owing to the small amounts of lead involved, certain factors not of any consequence in ordinary analysis, have to be considered:

1. In the isolation and estimation of lead by means of the formation of insoluble salts as precipitates the solubility factor is important. The most "insoluble" salts are soluble to a certain extent at great dilution, so that in any method the loss of lead must be reduced to a minimum by a precise knowledge of the optimum conditions for precipitation in all stages of the analysis.

2. Small amounts of lead are introduced in the reagents and apparatus employed during the course of the analysis. Although extremely small, they are relatively large when compared to the amount of lead actually present in the urine, hence, apart from the obvious necessity of using lead-free reagents, the number of steps in the analysis must be minimized.

Of these factors the loss of lead is more likely to be the source of error.

The various methods will now be briefly described. The details of the methods actually used in the investigation are given in Appendix I.

#### *1. Chemical Methods.*

A purely chemical method has been worked out by Avery, Hemingway and Anderson[3] after an extensive investigation. Briefly the method is as follows:

(*a*) The urine is evaporated to dryness and ashed with nitric acid.

(*b*) Lead (and allied metals if present) are separated from a hydrochloric acid solution of the ash by means of hydrogen sulphide.

(*c*) The sulphides are dissolved in nitric acid and the lead isolated as the sulphate.

(*d*) The lead sulphate is dissolved in ammonium acetate and estimated colorimetrically by means of the sulphide reaction.

*2. Physico-Chemical Methods.*

In these methods the lead is removed from the urine directly by making use of the phenomenon of adsorption. The small quantity of lead is adsorbed on the surface of a much larger precipitate of some other substance formed in the urine. It is then estimated by some characteristic chemical reaction.

Adsorption was first employed by Meillère[10] who worked out the following method for the detection of lead in urine:

(*a*) Copper sulphate is added to urine and hydrogen sulphide gas passed in.

(*b*) The lead is adsorbed on the copper sulphide precipitate and is separated from the copper by electrolysis.

(*c*) The anode is examined for lead by Trillat's reagent, a leuco base of tetra-methyl-diamino-diphenyl-methane.

Thresh[5] used the following method for quantitatively estimating lead in urine:

(*a*) The urine is made alkaline with sodium carbonate.

(*b*) The lead is adsorbed on the precipitated phosphates.

(*c*) The precipitate is dissolved in acetic acid.

(*d*) The lead is estimated colorimetrically by the sulphide reaction.

Fairhall[1] proposed the following:

(*a*) The urine is made alkaline with ammonia.

(*b*) The lead is adsorbed on the precipitated phosphates.

(*c*) The precipitate is ashed and the lead separated by means of hydrogen sulphide.

(*d*) The lead sulphide is converted to chromate.

(*e*) The lead is estimated by titrating the chromate with sodium thiosulphate solution.

Taylor's method[2] is as follows:

(*a*) The lead is adsorbed on the precipitate of calcium oxalate formed by adding ammonium oxalate and calcium chloride to the urine made acid with acetic acid.

(*b*) The precipitate is dissolved in hydrochloric acid and ashed.

(*c*) The lead is converted to hydroxide and then to chloride.

(*d*) The lead is estimated turbimetrically by means of the sulphite reaction (Ivanov's reaction).

*3. Electro-Chemical Methods.*

The conditions for the electrolytic determination of lead are not fully understood. A number of factors is involved which includes the acidity of the electrolyte, current strength, time of electrolysis, type of electrodes, influence of salts, among other things. A certain amount of information is available, but it is very conflicting.

From experimental work on the electrolysis of lead from nitric acid solutions, it has been repeatedly observed that lead is deposited both on the anode and the cathode, the proportion on each depending on the concentration of nitric acid.

Mellon and Reinhard[13] showed that with solutions of lead containing 4% and 8% free nitric acid, there is approximately equal distribution. As the concentration of nitric acid increases there is a tendency to form the peroxide until at a concentration of 20% nitric acid nearly all the lead is deposited in that form.

Schumm[9] obtained similar results and further showed that the presence of chlorides interfered with the deposition of lead. The electrolytic separation of lead from copper in 5% nitric acid solution in Meillère's method is considered by Schumm to be satisfactory.

In a method by Denis and Minot,[7] after ashing the urine and separating lead and allied metals as sulphides from an alkaline solution of the ash, the lead is isolated electrolytically in a 10% nitric acid solution. The method was later condemned by Minot[8] because manganese is also deposited with the lead and was estimated as lead.

So far no attempt has been made to isolate lead directly from urine by electrolysis. The question is mentioned by Avery, Hemingway and Anderson, but no details are given and they abandoned it for the chemical method. The writer carried out some experiments with a view to obtaining some information. To one litre of urine was added 0·1 milligramme of lead. The urine was evaporated to less than ninety cubic centimetres and nitric acid added so that the solution for electrolysis contained approximately 10% acid. The solution was electrolysed between flat platinum electrodes three by two centimetres at a current strength of six volts and 0·25 ampères. After twenty-four hours the electrodes were removed, the lead dissolved off in nitric acid and estimated quantitatively by the chemical method. The lead was also determined in the remaining electrolyte by the same method. The following results were obtained:

| Sample. | Milligramme of Lead. | | |
|---|---|---|---|
| | On Anode. | On Cathode. | Remaining Electrolyte. |
| A | 0·02 | 0·03 | 0·11 |
| B | 0·02 | 0·01 | 0·10 |
| C | 0·01 | Nil | 0·13 |

These results show the distribution of lead between anode and cathode under such conditions

of electrolysis and that (*a*) there is some factor present which prevents the deposition of lead; (*b*) the total amount of lead recovered is greater than the amount added, indicating that lead is normally present in urine in small amounts.

The same experiment was carried out on two urines from men who were suspected of having lead poisoning:

| Sample. | Quantity in Cubic Centimetres. | Milligramme of Lead. | | | |
|---|---|---|---|---|---|
| | | On Anode. | On Cathode. | Remaining Electrolyte. | Total Lead. |
| 1 | 790 | 0·05 | 0·05 | 0·04 | 0·14 |
| 2 | 950 | 0·02 | 0·15 | 0·06 | 0·23 |

It will thus be seen that further work is necessary in order to determine the right conditions for quantitative work. The electrolytic method was set aside as chemical methods proved to be satisfactory.

*Adsorption Methods.*

As the amount of lead to be estimated is very small it is of prime importance to know that all the lead is removed from the urine in the precipitate and none remains behind in the filtrate. The complete adsorption of lead depends on the physical character of the precipitate and this is governed by the reaction of the solution and the presence of other salts, both of which are variables in the case of urine. Experiments were carried out in which 0·1 milligramme lead (as nitrate) was added to five hundred cubic centimetres of urine and the procedure carried out according to the method being examined. After filtration the filtrate was examined for lead by the chemical method. It was found that where adsorption takes place in alkaline solution, the removal of lead is not complete and can be detected in the filtrate. There are thus decided objections to the procedure of adsorption as employed by Thresh and Fairhall. It was shown by Taylor[2] that as the alkalinity of the urine increases, the amount of lead adsorbed decreases and in a recent account of his method[14] the urine is adjusted to a definite hydrogen ion concentration before adsorption is carried out. The complete separation by adsorption of a known amount of a lead salt added to a urine does not constitute definite proof, as it is not known in what manner lead is combined with the urine. It is probably present in a highly dispersed state, but whether as a phosphate, as Fairhall suggests, or as part of an organic molecule, is uncertain. This constitutes an unknown factor in adsorption methods.

With Meillère's method it was found that the passing of hydrogen sulphide gas into a litre of urine was attended with mechanical difficulty and the subsequent filtration of the combined sulphide made the method very cumbersome. Separation of lead from copper is satisfactory, but the method is not quantitative.

Thresh's method has the further disadvantage that it does not eliminate the influence of copper in the final colorimetric determination.

The final estimation of the lead in Fairhall's method by means of a titration with $^{n}/_{200}$ sodium thiosulphate is unsuitable for the determination of lead in urine as it is not sufficiently sensitive. The final reaction is:

$$2PbCrO_4 + 16HCl + 6KI = 2PbCl_2 + 6KCl + 2CrCl_3 + 3I_2 + 8H_2O$$

from whence it can be calculated that one cubic centimetre of the standard solution = 0·345 milligramme lead which is too gross for the present purpose, but is excellent where large quantities of lead have to be determined as in fæces and tissues. The dilution of the standard solution was attempted in an endeavour to make the method more sensitive, but the solution was found to be very unstable.

In the method of Taylor lead may be lost at the ignition stage. The addition of hydrochloric acid converts all the calcium oxalate into chloride and also any lead present. Lead chloride is volatile at 600° C. which is about dull redness, hence unless very great precautions are taken, loss of lead will readily occur. When experiments were carried out, it was found difficult to avoid loss in this way. The estimation of the lead is carried out by means of a reaction due to Ivanov[15] in which lead forms a very insoluble white sulphite which causes an opalescence or turbidity in small amounts. The judgement of this opalescence when compared with that obtained from a known amount of lead constitutes the basis of the estimation. Precautions are taken to insure that those conditions which determine the physical character of the precipitate, that is, the reaction of the solution and the concentration of the salts, are the same in both unknown and standard.

The reaction of Ivanov is very sensitive and will enable the observer to detect one part of lead in 2,000,000. This is not, however, as sensitive as the well-known sulphide or Nessler reaction which will disclose one part of lead in 10,000,000. The reaction of Ivanov is given by other metals besides lead which occur in urine, for example, barium and tin. These were shown by Taylor to be eliminated during the analysis.

Copper, silver, nickel, iron, aluminium and calcium do not interfere. Manganese is not mentioned, although Reiman[12] showed this and zinc to be constituents of urine.

It is essential in an estimation of lead when the comparison of a colour or turbidity is made, that the colour or turbidity is given only by lead or, where it is also given by other metals, that lead only is present in the comparative stage.

The method of Avery, Hemingway and Anderson provides that only lead is present in the final comparative stage to give the colour reaction, all other metals having been eliminated in the various stages of the method.

*Chemical Methods.*

The only method available capable of accurately estimating small quantities of lead is that of Avery, Hemingway and Anderson. Examination of this method revealed the fact that accuracy was not attained without strict adherence to the conditions laid down at each stage by these workers. It was soon realized that loss of lead can occur at the stage of ignition in the incomplete precipitation of lead by hydrogen sulphide and in the isolation of lead as sulphate unless precautions are taken. Hence special attention was paid to these points.

There is no loss of lead on ignition provided the temperature is kept below 500° C. by the use of a well regulated furnace, preferably electric. Urine evaporated with nitric acid ashes readily and leaves a white ash completely soluble in dilute hydrochloric acid.

The complete precipitation of a small amount of lead in hydrochloric acid by means of hydrogen sulphide depends on (*a*) the reaction of the solution, (*b*) the influence of salts.

In the method of Avery small amounts of lead are precipitated from a hydrochloric acid solution approximately one-twentieth normal and found to be complete. The influence of other substances present in the ash of urine is not mentioned. At the stage of precipitation these are chiefly calcium, sodium and potassium chlorides and their influence was investigated. Mertens[16] observed that calcium chloride could prevent the precipitation of lead sulphide, but Hager and Geissler showed that this occurred when a large excess of hydrochloric acid was present.

Dede and Bonin[17] investigated the action of neutral chlorides, such as calcium chloride, ammonium chloride and potassium chloride on the precipitation of lead sulphide by hydrogen sulphide and found that, as the concentration of chlorides increased, the acidity of the solution must be decreased as under in order to bring about precipitation.

| Concentration of Chlorides. | Concentration of Hydrochloric Acid which will prevent Precipitation of Lead Sulphide. |
|---|---|
| 1·0 normal | 0·8 normal |
| 2·8 normal | 0·2 normal |

In a twenty-four hours' sample of urine the concentration of the chlorides is approximately ten grammes and by calculation the concentration of the chlorides present in the solution prior to precipitation with hydrogen sulphide is approximately normal and, as shown by Dede and Bonin, an acidity of 0·8 normal hydrochloric acid will prevent the precipitation of the lead. It will be seen, however, that the concentration of hydrochloric acid given by Avery, Hemingway and Anderson is very much smaller than this, namely 0·05 normal hydrochloric acid, and hence no loss of lead from incomplete precipitation occurs. If the acidity is reduced still further by making the solution acid to methyl orange (equivalent to 0·0001 normal hydrochloric acid), the greatest precaution of preventing loss will be taken.

The use of an added copper salt to assist in completely precipitating the lead was not found to have any advantage.

The loss of lead which occurs in the isolation of lead as sulphate, is the result of the slight solubility of lead sulphate in water. Although the authors have investigated this and sought to minimize it by the use of water-alcohol mixture, there remained an amount of lead of the order of 0·01 milligramme, which is constant and does not vary whatever the amount of lead present. Hence the loss of lead by this method should not be greater than 0·01 milligramme.

The lead sulphate is then dissolved in ammonium acetate and estimated colorimetrically. The colorimetric estimation of lead is carried out by means of the sulphide or Nessler reaction. This reaction is a very old one and with proper technique will lead to the detection of one part of lead in 10,000,000. The method of estimating lead is to compare the colour produced by adding ammonium sulphide to the unknown solution with that produced from a solution containing a known amount of lead.

It is carried out in alkaline solution in order to prevent the interference of traces of copper, bismuth, arsenic, antimony and tin. Methods involving the final comparison in acid solution have been suggested by some workers. Thresh[5] used an acetic acid solution, but did not remove the influence of copper. Siegfried and Pozzi[4] employed a nitric acid solution, but showed only that iron and manganese do not interfere. In both these methods use is made of a protective colloid, such as gum arabic or gelatine, in order to render visible and more stable the highly dispersed lead sulphide. The writer found that increased sensitiveness was obtained by this means, an amount of lead equivalent to 0·005 milligramme being easily detected. By experiment Bishop[18] showed that the most sensitive range for comparison was from 0·1 to 0·2 milligramme of lead.

**Preliminary Experimental Work.**

*Lead in Reagents and Apparatus.*

Lead must not be introduced in any considerable amount during the course of the analysis. Lead is sometimes found in the reagents or on the surface of the apparatus employed. In the case of the reagents the purest obtainable were employed;

| Reagent. | Amount used in the Analysis. |
|---|---|
| | Cubic Centimetres. |
| Nitric acid | 50 |
| Hydrochloric acid | 5 |
| Sulphuric acid | 1 |
| Ammonium hydroxide | 1 |
| Ammonium acetate, 30% | 10 |
| Potassium cyanide, 10% | 1 |
| Alcohol, 95% | 10 |
| Water | 250 |

further, the nitric acid, hydrochloric acid, alcohol and water were redistilled to render them lead-free, the remaining reagents requiring no special treatment, as they were found to be free from detectable lead in the quantities used:

In order to determine whether any lead was introduced by the use of porcelain, silica and glassware during the course of the analysis, the method was carried out in its entirety on one litre amounts of lead-free distilled water and it was found that the amount of lead taken up in this way was not greater than 0·02 milligramme.

The presence of small amounts of lead in filter papers was observed by Avery, Hemingway and Anderson and this was found to be the case in the present instance. The filter papers were rendered lead free by washing with hot dilute hydrochloric acid, followed by ammonium acetate.

*Lead in Normal Urine.*

The estimation of the lead present in the urine of five persons (medical students) with no exposure to lead was carried out on one litre samples by the chemical method, a blank experiment with distilled water being conducted at the same time.

The following results were obtained.

Blank experiment = 0·015 milligramme of lead.

| Lead Found, Milligramme. | Net Lead, Milligramme. |
|---|---|
| 0·05 | 0·035 |
| 0·06 | 0·045 |
| 0·065 | 0·05 |
| 0·07 | 0·055 |
| 0·06 | 0·045 |

There is thus an average excretion of 0·04 milligramme lead per litre in persons not exposed to lead. This is probably obtained from the food.

Analyses of the food of a hospital patient on three successive days gave the following results:

| Date. | Weight of Food, Grammes. | Lead, Milligrammes. |
|---|---|---|
| 11.1.28 | 926 | 1·15 |
| 12.1.28 | 793 | 0·14 |
| 13.1.28 | 1,218 | 0·25 |

*Recovery of Added Lead.*

Two litres of normal urine were divided into four portions of five hundred cubic centimetres and to three portions were added known amounts of lead. The lead was estimated in all the samples. A blank experiment was carried out at the same time. The following results were obtained after deducting the blank:

| Lead Added, Milligramme. | Lead Found, Milligramme. | Percentage Recovery. |
|---|---|---|
| Nil | 0·02 | — |
| 0·05 | 0·06 | 80 |
| 0·10 | 0·115 | 95 |
| 0·15 | 0·16 | 93 |

A further check was made by dividing a twenty-four hours' specimen of urine from a man with a history of exposure to lead into two unequal portions and estimating the lead in each.

The total sample comprised 1,100 cubic centimetres. The amount of lead found in four hundred and fifty cubic centimetres was 0·14 milligramme and in six hundred and fifty cubic centimetres 0·21 milligramme. There was therefore 0·35 milligramme of lead in the whole sample.

It will be seen that the results are proportional to the amount of sample taken for analysis.

In the present investigation the estimation of lead was carried out by the chemical method of Avery, Hemingway and Anderson owing to the many uncertainties in the other methods examined. As the state of combination of lead in the urine of men exposed to lead is not known, it was considered wiser to employ a method which would not only overcome this difficulty but was equally accurate and reliable. The chemical method has at each stage of the process well-defined conditions for optimum separation of the lead. Both the gain and loss of lead are of small magnitude and can be allowed for. The final colorimetric estimation is free from all interfering substances and is very sensitive (one part of lead in 10,000,000). Finally the method can be used for the determination of lead in all classes of biological material by simple changes in the procedure.

It was, therefore, admirably suitable for the present investigation and all determinations of lead in urine have been carried out by this method.

*The Estimation of Lead in Fæces.*

The quantity of lead in the fæces of men exposed to lead is of such magnitude as to allow accurate estimation by volumetric methods and a modification of the method of Fairhall was found to be suitable.

The fæces were weighed, dried in an air oven at 110° C. and ashed in a silica dish at a low red heat. Fæces ash readily and require no addition of oxidizing agents as sodium nitrate or nitric acid. The ash was dissolved in dilute hydrochloric acid, transferred to a three hundred cubic centimetres Erlenmeyer flask and diluted to three hundred cubic centimetres with distilled water. The acidity was now adjusted until just acid to methyl orange by cautious addition of 10% sodium hydroxide. Hydrogen sulphide gas was then passed in for a quarter of an hour which precipitated the lead (and other metals of the same group, if present), the flask was stoppered and allowed to stand over night. The sulphide precipitate was filtered off, washed with hydrogen sulphide water. It was then dissolved in hot 1:1 nitric acid and the filter paper washed with hot distilled water. After neutralizing the solution with ammonium hydroxide and then acidfying with acetic acid, the lead was precipitated as chromate by the addition of a few drops of a saturated solution of potassium chromate. The solution was warmed to insure complete precipitation and the precipitate filtered off. It was thoroughly washed

with distilled water until free from potassium chromate, and then dissolved off by the addition of a few cubic centimetres of hydrochloric acid. The lead chromate in the solution was titrated with $^{n}/_{200}$ sodium thiosulphate, after the addition of a few crystals of potassium iodide, starch solution being used as an indicator. From the equation previously given, 1 c.c. $^{n}/_{200}$ $Na_2S_2O_3$ = 0·345 milligramme lead.

The method proved very satisfactory and the only complication was found when the fæces contained large amounts of bismuth, used in medication. As this metal belongs to the same group as lead, it is precipitated as a black sulphide, which is liable to be mistaken for lead. Its presence can usually be detected when the hydrochloric acid solution of the ash is diluted with water, the bismuth being precipitated as the white bismuth oxychloride. When bismuth occurs in the fæces the amount is very large compared to the amount of lead present. In one case the relative amounts were as follows: in one hundred grammes of material there were 2·47 grammes of bismuth and 2·54 milligrammes of lead.

It was realized that the complete separation of two closely allied metals, especially when one is in such large amounts compared to the other, was uncertain. Hence lead estimations in such cases are of doubtful accuracy and a fresh sample should be obtained after medication has ceased.

*The Estimation of Lead in Tissues.*

The estimation of lead in the various organs of the body, as in *post mortem* material, was carried out in a similar way to that of fæces, with the use of the chromate method. It was found that this organic material was not easy to ash, due largely to the fact that the material was not thoroughly dry. If the portions for analysis are chopped up, spread out and dried for two or three days at 110° C., ashing can readily be carried out at low red heat in a muffle furnace. Certain tissues ash more readily than others. Bone and liver ash readily, lung, kidney and spleen less readily, while muscular tissue, as heart and skeletal muscle, is more difficult. The use of an oxidizing agent as sodium nitrate was found necessary in most cases in order to oxidize the carbon remaining. The ash was then extracted with concentrated hydrochloric acid and placed on the water bath for two hours. After the addition of hot water it was decanted off. A second extraction was then made as before followed by filtration and the residue washed till free from chloride. The filtrate was evaporated to dryness on the water bath, diluted to two hundred cubic centimetres, the solution made just acid to methyl orange and hydrogen sulphide gas passed in. The subsequent stages were then carried out as in the case of fæces; the estimation of the lead was conducted by titration as the chromate.

A method proposed by Froboese[11] is intended to be for general application. It is a combination of various methods already discussed and, as it contained many unnecessary steps, does not possess any advantages over the methods adopted.

**Summary.**

1. A critical survey of the available methods for the determination of lead in biological material was carried out.
2. For the determination of small quantities of lead in urine the chemical method of Avery, Hemingway and Anderson was adopted.
3. For the estimation of larger quantities of lead in tissues and fæces, the volumetric method of Fairhall was found to be most suitable.

## APPENDIX I.

### METHODS FOR THE DETERMINATION OF SMALL AMOUNTS OF LEAD IN ORGANIC MATERIAL.

#### Determination of Lead in Urine.[3]

*Evaporation of the Urine.*

Place five hundred cubic centimetres of the sample in a seven inch porcelain basin (S.C.P. ware), add fifty cubic centimetres of nitric acid and evaporate on the hot plate, gradually adding another five hundred cubic centimetres of the sample, making one litre in all. Continue the evaporation until about thirty cubic centimetres are left.

*Destruction of Organic Matter.*

Transfer to a silica dish (about nine centimetres (three and a half inches) in diameter), cleaning out the porcelain basin with ten cubic centimetres of nitric acid and finally washing it with distilled water. Allow the contents of the dish to evaporate slowly on the hot plate to avoid loss by spraying. Continue the evaporation to dryness, being careful to avoid overheating which causes the mass to fume strongly and possibly to deflagrate. When dry, place the dish in a small muffle, preferably electrically heated, and heat until fuming ceases and the mass becomes charred, proceeding very cautiously to prevent deflagration, especially at first. Gradually increase the temperature by pushing the dish farther into the muffle until the carbon burns off quietly and the mass becomes white. Heat to between 450° and 500° C. until red fumes cease to be given off, usually in about twenty minutes. If the nitrates are not completely decomposed at this stage, the analysis will have to be rejected.

*Separation of Second-Group Metals.*

Cool, add hot water until the dish is about two-thirds full; heat to boiling on the hot plate, break up lumps with a glass rod and carefully add hydrochloric acid until the solution is clear and either neutral or very slightly acid. Then add exactly two cubic centimetres of six times normal hydrochloric acid in excess. Special attention must be paid to the acidity of the solution at this stage. The white residue after ignition is usually alkaline and must be carefully neutralized; then the required excess of acid is added. Filter off any small residue of silica through a nine centimetre filter paper, wash well with hot water, catching the filtrate in a three hundred cubic centimetre Erlenmeyer flask. Cool and make up the bulk to two hundred and fifty cubic centimetres. Fit the flask with a rubber stopper with two holes, through one of which a

straight tube passes nearly to the bottom of the flask, the end of this tube being drawn out to a fine jet. Through the other hole is passed another glass tube bent at right angles, going through the stopper for a length of about twelve millimetres (half an inch). Connect up with the hydrogen sulphide apparatus and bubble a slow stream of the gas through the cold solution for one hour. Allow to stand overnight, when any precipitate will settle out. Filter through a nine centimetre filter paper, wash out the flask three times with a small quantity of cold hydrogen sulphide water and then wash the filter paper three times with the same liquid.

*Separation of the Lead as Sulphate.*

Dissolve the precipitate of sulphides by dropping two cubic centimetres of hot 1:1 nitric acid from a pipette around the filter paper, followed by another two cubic centimetres of the same, catching the solution in the original Erlenmeyer flask. Wash six times with hot distilled water. Warm the solution in the flask and evaporate to about twenty cubic centimetres, then transfer to fifty cubic centimetre cylindrical beaker (non-alkali glass), washing out the flask three times with warm water. Evaporate quietly to about five cubic centimetres on the hot plate, then add one cubic centimetre of sulphuric acid and heat until strong fumes arise. Cool and add twenty cubic centimetres of cold water, then ten cubic centimetres of alcohol; mix well and allow to stand overnight. Filter through a nine-centimetre filter paper, washing out the beaker with a solution of sixty-five volumes water, thirty-two volumes of absolute alcohol and three volumes of sulphuric acid. Then wash the filter paper three times with the same solution.

*Colorimetric Determination of the Lead.*

Dissolve any precipitate off the filter paper by dropping five cubic centimetres of hot (1:2) ammonium acetate solution from a pipette around the filter paper, followed by another five cubic centimetres of the same reagent, catching the solution in the original fifty cubic centimetre beaker. Wash six times with hot water. Cool and Nesslerize, using a standard solution of lead acetate of 0·00001 gramme of lead in one cubic centimetre.

The Nesslerizing is carried out in the following manner:

Transfer the assay solution to a fifty cubic centimetre Nessler tube, add one cubic centimetre of a 10% potassium cyanide solution, one cubic centimetre of ammonium hydroxide and six drops of freshly prepared ammonium sulphide. Make up to the fifty cubic centimetre mark and mix well. This assay solution must be quite colourless. Add to it the standard lead acetate solution, stirring after each addition until the colour matches that of the assay, when both tubes are placed in a colorimeter. It is advisable to arrange the strength of the assay solution so that not more than eight cubic centimetres of the standard lead solution will be required for Nesslerizing. If much lead sulphate precipitate is noticed after the addition of water and alcohol, the solution in ammonium acetate must be made up to a definite volume and an aliquot part taken for Nesslerizing, so that not more than eight cubic centimetres of standard solution will be used in the comparison. In this case use the same proportion of (1:2) ammonium acetate for making up the comparison solution as is contained in the aliquot part of the assay solution. It is desirable in all cases to regard the first comparison as a trial and to make up another standard in which the required lead acetate solution is added before the ammonium sulphide and along with the ammonium acetate, potassium cyanide, ammonia and water to make up a total of fifty cubic centimetres. The solution is mixed well and 0·3 cubic centimetre of ammonium sulphide added and after further mixing compared with the assay. Slight additions may then be made to either assay or standard in order to match them and the necessary corrections made.

**The Chromate Method for the Determination of Lead.**[1]

*Ashing.*

Before analysis tissues and fæces must be freed from water by baking. This may be done very rapidly by heating the material in porcelain dishes on a hot plate until it starts to char. Transfer to the muffle furnace and ash to a dull red heat. Fæcal matter usually ashes readily, but the tissues form a residue which must be repeatedly extracted before the entire char is consumed. Usually most material requires re-ashing as a certain quantity of inorganic salts becomes fused and prevents complete oxidation. After the first ashing, the material should be cooled and extracted with dilute hydrochloric acid and hot water. It is essential that at this stage all the ash be dissolved, for frequently lead phosphate is present as an insoluble residue that might be mistaken for silica. If this residue is insoluble in hydrochloric acid, it should be treated with a mixture of hydrochloric and tartaric acids (which dissolves lead phosphate) until the ash is quantitatively dissolved.

In analysing urine for lead, the procedure has been to evaporate to dryness, char and ash the residue. Since urine residues are difficult to ash, because of the large quantity of inorganic salts present, repeated extraction and ashing is necessary if all the salts are to be dissolved. This makes the process burdensome. The following new method in which evaporation is avoided by precipitating lead directly from the urine, has been devised.

*Entrainment.*

Ammonium hydroxide is added to urine until it is strongly ammoniacal. The mixture is allowed to stand from one to twenty-four hours. In this reaction the earthy phosphates are precipitated and lead phosphate is carried down quantitatively by entrainment. The gelatinous mass of phosphates settles into a compact mass from which the clear lead-free liquor can be decanted and the remainder filtered by suction on a Buchner funnel. The filter paper containing the precipitate ashes readily in

a few minutes and the quantity of lead may be determined by the chomate method as follows.

*Precipitation.*

The strongly acid solution should be neutralized with sodium hydroxide. Hydrochloric acid should be added until the solution is just acid to methyl orange. Saturate the cold solution with hydrogen sulphide; if sulphides precipitate to any great extent during the process, they may be filtered at once, but if no precipitate appears, the solution, saturated with hydrogen sulphide, should be allowed to stand over night before filtration. Immediately after filtration the precipitate should be washed, as lead sulphide oxidizes rapidly when in contact with air. Solution of the washed precipitate in nitric acid, boiling to expel hydrogen sulphide, cooling and finally neutralizing with sodium hydroxide, as indicated by phenolphthalein, are then necessary. After reacidification with acetic acid, two or three drops of a saturated solution of potassium chomate should be added. To hasten the reaction the solution should be boiled for a few minutes. If no turbidity is present, the solution should stand over night before filtration to allow separation of the extremely small amounts of lead it may contain. After filtering all trace of soluble chromate should be washed from the filter paper.

*Titration.*

The chromate is dissolved in dilute hydrochloric acid, an excess of potassium iodide added at once and the free iodine titrated with 0·005 normal thiosulphate solution, starch being used as an indicator.

1 c.cm. = 0·3451 mg. Pb.

$$2PbCrO_4 + HCl + 6KI = 2PbCl_2 + 6KCl + 2CrCl_3 + 3I_2 + 8H_2O.$$

$$I_2 + 2Na_2S_2O_3 = 2NaI + Na_2S_4O_6.$$

## THE EXCRETION OF LEAD BY MINE WORKERS AT BROKEN HILL.

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The subjects of the investigation of the excretion of lead were men employed in the various processes concerned in the production of refined lead from lead ore. The mining operations are carried out at Broken Hill, New South Wales, and the production of refined lead at Port Pirie, South Australia. Owing to the different conditions existing at the two places, the subjects are considered separately as the Broken Hill group and the Port Pirie group.

*Mining Operations.*

The Broken Hill lode is complex in composition and is principally worked for lead and zinc. Besides containing these metals in the form of galena and blende respectively, the ore has a large proportion of valueless material or gangue. The composition of the ore varies in different parts of the lode as shown in Table I.(1)

The amount of galena (lead sulphide) in the ore varies from 12·76% to 18·38% and blende (zinc sulphide) from 19·2% to 29·2%. The mining operations are concerned with the extraction, crushing and concentration of the ore which is then sent to Port Pirie for refining. Before proceeding further certain definitions relating to the men examined are necessary.

Mine worker is a general term including all men employed in or about the mines, that is, on the surface or underground.

Surface worker includes men such as fitters, millhands, labourers and others whose work is confined to the surface.

Underground worker includes miners, truckers, mullockers and those whose industrial life is spent underground.

*Description of the Subjects.*

The subjects of this investigation were obtained from the following sources:

1. Applicants for employment at the Broken Hill Mines. This includes men who had not previously worked at the mines and also mine workers returning to work after an absence of at least six months.
2. Patients at the Broken Hill Hospital who were mine workers. They were mostly out-patients, but included a number of in-patients. These men were selected after medical examination to exclude any abnormality which would render them unsuitable for this investigation.

*Collection of Specimens.*

A specimen of urine was obtained from applicants for employment and hospital out-patients.

Specimens of urine and fæces were obtained from the in-patients at intervals, each specimen representing the total output for the day. Full particulars of the subjects' industrial history were also recorded.

TABLE I.
MINERALOGICAL PERCENTAGE COMPOSITION OF BROKEN HILL MINE ORES.

| Mineral. | North. | Junction North. | British. | Block 14. | B.H.P. | Block 10. | Central. | South. | Zinc. |
|---|---|---|---|---|---|---|---|---|---|
| Free silica .. .. .. | 15·16 | 1·62 | 15·2 | 11·74 | 12·0 | 7·01 | 12·24 | 17·73 | 17·40 |
| Silicates.. .. .. .. | 39·57 | 66·41 | 48·77 | 47·43 | 46·28 | 57·25 | 37·81 | 24·37 | 33·28 |
| Galena .. .. .. .. | 17·69 | 14·34 | 13·69 | 16·88 | 14·64 | 12·76 | 16·87 | 18·38 | 18·21 |
| Blende .. .. .. .. | 22·03 | 14·05 | 19·64 | 21·48 | 23·74 | 20·07 | 29·2 | 26·95 | 19·92 |
| Sulphides of copper .. .. | 0·22 | 0·25 | 0·22 | 0·22 | 0·19 | 0·22 | 0·22 | 0·21 | 0·27 |
| Sulphides of arsenic and antimony .. .. .. | 0·09 | 0·07 | 0·10 | — | 0·07 | 0·09 | — | 0·10 | 0·12 |
| Calcium carbonate .. .. | 5·24 | 3·02 | 1·7 | 2·57 | 2·73 | 2·60 | 3·79 | 11·15 | 10·80 |
| Calcium fluoride .. .. | — | 0·2 | 0·19 | — | — | — | 0·22 | 0·39 | — |
| Pyrite .. .. .. .. | — | 0·04 | 0·21 | — | 0·15 | — | 6·30 | 0·32 | — |
| Pyrrhotite .. .. .. | — | — | 0·28 | — | 0·20 | — | — | 0·40 | — |

*Conditions of Exposure to Lead.*

The majority of the men examined were underground workers and the conditions under which these men work, were fully investigated by the Technical Commission of Inquiry, 1919-1921. The commission found that the men were exposed to the action of dust containing lead which enters the body by way of the respiratory tract and to a lesser extent by the alimentary tract. From analyses of the dust present after blasting operations in the mines it was concluded that the main constituents of the ore were also those of the dust and, although the minerals composing the ore differ greatly in hardness and other physical properties, the dust is fine enough to remain suspended in the air for some time, as shown in the Tables II and III.

TABLE II.
COMPOSITION OF DUST OF ORE FROM WHICH IT WAS OBTAINED BY BLASTING.

| Amount of Material. | Stope. | | | |
|---|---|---|---|---|
| | Calcitic. | Siliceous. | Rhodonitic. | Oxidized. |
| Weight of dust .. | 461 mg. | 217 mg. | 25 mg. | 17 mg. |
| Dust, insoluble .. | 7·25% | 13·36% | 44·2% | 44·18% |
| Ore, insoluble .. | 13·54% | 47·04% | 44·0% | 60·92% |
| Dust lead .. .. | 35·66% | 34·59% | 7·50% | 5·88% |
| Ore lead .. .. | 31·68% | 20·14% | 10·02% | 7·36% |
| Dust zinc .. .. | 15·20% | 25·07% | — | — |
| Ore zinc .. .. | 15·89% | 15·48% | 12·79% | — |

TABLE III.
EFFECT OF PERIOD OF SETTLING ON COMPOSITION OF DUST.

| Dust from Calcitic Stope. | First 45 minutes' collection. | Second 40 minutes' collection. |
|---|---|---|
| Weight of sample .. .. .. | 386 mg. | 75 mg. |
| Insoluble .. .. .. .. | 7·25% | 10·66% |
| Lead .. .. .. .. .. | 35·66% | 22·32% |
| Zinc .. .. .. .. .. | 15·20% | 20·64% |
| Oxides of iron, aluminium and manganese .. .. .. | 7·04% | 9·60% |
| Lime (CaO) .. .. .. .. | 7·07% | 10·40% |

It will be seen from Table II that the composition of the dust obtained by blasting is not the same as the ore mined and that the percentage of lead in the dust may be greater or less than the percentage in the ore. It is most increased in the dust from siliceous ores. Thus underground workers especially miners are often exposed to the action of lead in the form of lead sulphide in high concentrations in the dust of the air breathed. Although surface workers are not under the same conditions as the underground workers, there are in the processes of handling and crushing the ore many opportunities of exposure to lead, but no information is available of the dust conditions at the various stages of these operations.

**Lead Excretion of all Subjects.**

In Table IV the result of determinations of the amount of lead excreted in the urine of all the subjects is given, together with related information. In each case the amount in the specimen received and for purposes of comparison the amount per litre are recorded. It will be seen that only in a few cases was the twenty-four hours' urine obtained. As a result of the smallness of the sample in certain cases lead was not detected and is shown as nil in the table, although the subject may be excreting small amounts of lead. (Owing to the sensitiveness of the method these amounts would not be greater than 0·02 milligramme per litre.) All estimations were carried out by the method of Avery, Hemingway and Anderson as previously described.

The results show that the lead excretion in the urine of all subjects at Broken Hill varies from an extremely small amount to as much as 0·5 milligramme per litre. The distribution is shown in Table IV.

TABLE IV.
LEAD EXCRETION.

| Milligramme of Lead per Litre of Urine. | Number of Persons. |
|---|---|
| 0·05 or less | 36 |
| 0·06 to 0·09 | 11 |
| 0·10 to 0·19 | 29 |
| 0·20 to 0·29 | 14 |
| 0·30 to 0·39 | 6 |
| 0·40 to 0·49 | 4 |
| | 100 |

**The Relation of the Period of Exposure to the Excretion of Lead in Urine.**

It will be seen from Table V that the subjects are men of many nationalities, of various ages, with periods of employment in the industry ranging from one month to over twenty years, during which time the subject has been exposed more or less to the action of lead. The total period of employment represents the whole of the subject's life as a mine worker and may be the sum of a number of broken periods of the same kind of mining work or it may represent a period of surface work followed by underground work.

TABLE V.
THE LEAD EXCRETION OF ALL SUBJECTS AT BROKEN HILL.

| No. | Age, Years. | Nationality. | Nature of Work. | Total Period Employment at the Mines in Years. | Lead Excretion in Urine. | | |
|---|---|---|---|---|---|---|---|
| | | | | | Quantity of Specimen in Cubic Centimetres. | Total Lead in Specimen, Milligramme. | Lead per Litre, Milligramme. |
| 4 | 33 | Maltese | Miner | 1·33 | — | Nil | — |
| 7 | 21 | Australian | — | Nil | 430 | 0·04 | 0·09 |
| 8 | 19 | Australian | Labourer | 1·0 | 570 | 0·05 | 0·09 |
| 9 | 26 | Jugo-Slav | — | Nil | 420 | 0·01 | 0·03 |
| 10 | 27 | Australian | Mullocker | 1·0 | 560 | Nil | — |
| 11 | 20 | Australian | — | Nil | 600 | 0·12 | 0·19 |
| 12 | 25 | Greek | — | Nil | 330 | 0·01 | 0·03 |
| 13 | 25 | Jugo-Slav | — | Nil | 450 | Nil | — |
| 14 | 15 | Australian | — | Nil | 660 | 0·02 | 0·03 |
| 15 | 29 | Australian | Miner | 3·5 | 540 | 0·03 | 0·05 |
| 16 | 31 | Greek | — | Nil | 175 | 0·01 | 0·06 |
| 17 | 22 | Australian | Various | 2·5 | 375 | 0·03 | 0·08 |
| 18 | 49 | Australian | Timber man | 1·9 | 440 | 0·01 | 0·02 |
| 19 | 24 | Australian | — | Nil | 550 | 0·04 | 0·08 |
| 20 | 34 | Australian | Miner | 12·0 | 380 | Nil | — |
| 21 | 22 | Australian | Trucker | 0·25 | 440 | 0·035 | 0·08 |
| 22 | 35 | Australian | Mullocker | 13·25 | 660 | Nil | — |
| 23 | 45 | Australian | Miner | 1·0 | 440 | 0·02 | 0·05 |
| 24 | 35 | English | Miner | 5·66 | 400 | 0·03 | 0·08 |

TABLE V (*Continued*).

| No. | Age, Years. | Nationality. | Nature of Work. | Total Period Employment at the Mines in Years. | Lead Excretion in Urine. | | |
|---|---|---|---|---|---|---|---|
| | | | | | Quantity of Specimen in Cubic Centimetres. | Total Lead in Specimen, Milligramme. | Lead per Litre, Milligramme. |
| 25 | 21 | Australian | Trucker | 0·9 | 550 | 0·06 | 0·11 |
| 26 | 30 | Bulgarian | Mullocker | 4·0 | 570 | 0·09 | 0·16 |
| 27 | 35 | Australian | — | Nil | 410 | 0·01 | 0·02 |
| 28 | 19 | Australian | Labourer | 0·25 | 360 | 0·10 | 0·27 |
| 29 | 26 | Maltese | Miner | 2·33 | 420 | Nil | — |
| 30 | 34 | Maltese | Miner | 3·5 | 510 | Nil | — |
| 31 | 34 | Maltese | Miner | 1·16 | 450 | 0·09 | 0·20 |
| 32 | 27 | Maltese | — | Nil | 470 | 0·04 | 0·08 |
| 33 | 35 | Australian | Miner | 12·5 | 430 | Nil | — |
| 34 | 22 | Maltese | — | Nil | 370 | Nil | — |
| 35 | 26 | Jugo-Slav | — | Nil | 450 | 0·05 | 0·12 |
| 36 | 28 | Italian | — | Nil | 430 | Nil | — |
| 37 | 22 | Australian | Labourer | 4·9 | 500 | 0·11 | 0·22 |
| 38 | 38 | Italian | Miner | 5·0 | 430 | 0·05 | 0·11 |
| 39 | 44 | Italian | Miner | 1·5 | 460 | 0·08 | 0·17 |
| 40 | 29 | Australian | Miner | 0·41 | 450 | Nil | — |
| 41 | 28 | Jugo-Slav | Labourer | 0·58 | 660 | 0·02 | 0·03 |
| 42 | 38 | Italian | Mullocker | 1·0 | 560 | 0·05 | 0·09 |
| 43 | 21 | South African | Miner | 3·33 | 390 | 0·11 | 0·29 |
| 44 | 22 | Australian | Labourer | 1·5 | 520 | 0·10 | 0·19 |
| 45 | 41 | Jugo-Slav | Miner | 9·75 | 360 | 0·05 | 0·19 |
| 46 | 25 | Greek | — | Nil | 250 | 0·06 | 0·25 |
| 47 | — | — | — | — | 575 | 0·10 | 0·17 |
| 48 | 34 | Scotch | — | Nil | 390 | 0·07 | 0·18 |
| 50 | 53 | Australian | Miner | 25·75 | 1,100 | 0·35 | 0·32 |
| 51 | 23 | Jugo-Slav | Trucker | 0·58 | 540 | 0·10 | 0·18 |
| 52 | 24 | English | Trucker | 0·33 | 430 | 0·16 | 0·37 |
| 53 | 25 | Maltese | Trucker | 2·66 | 310 | 0·02 | 0·06 |
| 54 | 43 | Australian | Miner | 14·83 | 300 | 0·06 | 0·20 |
| 55 | 24 | Australian | Trucker | 1·75 | 530 | 0·11 | 0·20 |
| 56 | 41 | Maltese | Trucker | 3·33 | 500 | 0·19 | 0·38 |
| 57 | 46 | Indian | Trucker | 1·5 | 570 | Nil | — |
| 58 | 28 | Greek | Trucker | 2·83 | 380 | 0·05 | 0·13 |
| 59 | 33 | Italian | Trucker | 1·41 | 420 | 0·02 | 0·05 |
| 60 | 21 | Australian | Miner | 2·66 | 1,350 | 0·47 | 0·36 |
| 62 | 44 | Italian | Trucker | 1·0 | 650 | 0·07 | 0·11 |
| 63 | 30 | Italian | Trucker | 0·5 | 670 | 0·09 | 0·13 |
| 64 | 26 | Jugo-Slav | Trucker | 1·83 | 690 | 0·10 | 0·14 |
| 65 | 28 | Jugo-Slav | Trucker | 2·25 | 540 | 0·09 | 0·17 |
| 66 | 36 | Irish | Miner | 4·91 | 320 | 0·09 | 0·28 |
| 67 | 29 | Jugo-Slav | Trucker | 0·75 | 345 | 0·17 | 0·49 |
| 68 | 55 | Jugo-Slav | Filler | 0·08 | 725 | 0·11 | 0·15 |
| 69 | 27 | Maltese | Trucker | 0·58 | 460 | 0·06 | 0·13 |
| 70 | 35 | Italian | Miner | 3·66 | 1,450 | Nil | — |
| 71 | 32 | New Zealand | Miner | 0·75 | 1,280 | 0·22 | 0·17 |
| 72 | — | — | Miner | — | 760 | 0·14 | 0·18 |
| 73 | 41 | Italian | Miner | 1·41 | 475 | 0·01 | 0·02 |
| 74 | 21 | English | Trucker | 0·66 | 275 | Nil | — |
| 75 | 34 | Italian | Miner | 2·16 | 480 | 0·01 | 0·02 |
| 76 | 28 | Maltese | Trucker | 0·75 | 530 | 0·05 | 0·09 |
| 77 | 34 | Greek | Miner | 0·5 | 400 | 0·02 | 0·05 |
| 78 | 37 | Australian | Miner | 6·0 | 350 | 0·01 | 0·03 |
| 79 | 29 | Greek | Miner | 2·91 | 700 | 0·01 | 0·01 |
| 80 | 35 | Australian | Miner | 10·16 | 320 | 0·06 | 0·18 |
| 81 | 35 | Greek | Miner | 2·0 | 615 | 0·07 | 0·11 |
| 82 | 43 | Australian | Miner | 8·83 | 680 | 0·11 | 0·16 |
| 83 | 43 | Australian | Trucker | 2·5 | 310 | Nil | — |
| 84 | 22 | Australian | Open Cut | 2·41 | 350 | Nil | — |
| 85 | 31 | Maltese | Miner | 8·33 | 660 | 0·03 | 0·04 |
| 86 | 35 | Australian | Miner | 10·25 | 525 | 0·01 | 0·02 |
| 87 | 33 | Australian | Millhand | 13·5 | 435 | 0·01 | 0·02 |
| 88 | 36 | Italian | Miner | 2·5 | 360 | 0·14 | 0·39 |
| 89 | 30 | Jugo-Slav | Mullocker | 2·66 | 60 | 0·01 | 0·16 |
| 90 | 36 | Australian | Miner | 12·58 | 220 | 0·06 | 0·27 |
| 91 | 38 | Australian | Miner | 9·16 | 480 | 0·12 | 0·25 |
| 92 | 30 | Jugo-Slav | Miner | 3·0 | 190 | 0·02 | 0·10 |
| 93 | 39 | Australian | Miner | 14·0 | 250 | 0·05 | 0·20 |
| 94 | 34 | Greek | Trucker | 0·91 | 170 | 0·05 | 0·29 |
| 95 | 31 | Italian | Miner | 3·08 | 225 | 0·10 | 0·44 |
| 96 | 27 | Jugo-Slav | Miner | 2·83 | 195 | 0·05 | 0·25 |
| 97 | 39 | Jugo-Slav | Miner | 3·0 | 135 | 0·02 | 0·15 |
| 98 | 25 | Jugo-Slav | Miner | 3·0 | 660 | 0·09 | 0·13 |
| 100 | 43 | Australian | Miner | 10·0 | 1,860 | 0·42 | 0·22 |
| 101 | 40 | Australian | Miner | 5·0 | 1,200 | 0·44 | 0·37 |
| 103 | 42 | Italian | Miner | 0·08 | 80 | Nil | — |
| 105 | 29 | Jugo-Slav | Trucker | 2·0 | 235 | 0·10 | 0·42 |
| 106 | 26 | Norwegian | Miner | 0·91 | 190 | 0·09 | 0·47 |
| 107 | 35 | Italian | Miner | 1·0 | 315 | 0·06 | 0·19 |
| 108 | 32 | Italian | Miner | 1·66 | 1,540 | 0·04 | 0·03 |
| 109 | 52 | English | Miner | 21·23 | 1,550 | 0·17 | 0·11 |
| 110 | 30 | Swedish | Miner | 1·66 | 1,530 | 0·04 | 0·03 |

A careful analysis of the information contained in the table was therefore carried out. A first consideration was to find out the so-called normal excretion of lead, that is, the excretion of lead by men who have never been exposed to the conditions existing in the mines, but who, nevertheless, have lived at Broken Hill for various periods.

*The Excretion of Lead by Men who have Never Been Exposed to Lead.*

Among the men examined were many proceeding to mining work and giving no history of previous exposure to lead. The lead excretion of this group is shown in Table VI and serves as a control or as a standard when comparing the effect of the period of exposure on the urinary lead excretion of mine workers.

TABLE VI.

THE URINARY LEAD EXCRETION OF MEN AT BROKEN HILL WHO HAVE NOT BEEN EXPOSED TO LEAD.

| No. | Age. | Nationality. | Lead in Urine, Milligramme per Litre. | Length of Residence in Broken Hill. |
|---|---|---|---|---|
| 7 | 21 | Australian | 0·09 | Two weeks |
| 9 | 26 | Jugo-Slav | 0·03 | Seven weeks |
| 11 | 20 | Australian | 0·19 | Born at Broken Hill, nine months recent |
| 12 | 25 | Greek | 0·03 | Five months |
| 13 | 25 | Jugo-Slav | <0·02 | Two months |
| 14 | 15 | Australian | 0·03 | Born at Broken Hill, whole life |
| 16 | 31 | Greek | 0·06 | Two months |
| 19 | 24 | Australian | 0·08 | One week |
| 27 | 35 | Australian | 0·02 | — |
| 32 | 27 | Maltese | 0·08 | — |
| 34 | 22 | Maltese | <0·02 | Three months |
| 35 | 26 | Jugo-Slav | 0·12 | Two months |
| 36 | 28 | Italian | <0·02 | One month |
| 46 | 25 | Greek | 0·25 | — |
| 48 | 34 | Scotch | 0·18 | — |

With respect to the high lead excretions of Numbers 11, 35, 46 and 48 in Table VI, the clinical record cards have been carefully examined. Two of them, Numbers 11 and 35, had been residing for some time in Broken Hill and may, therefore, have absorbed lead in drinking water. No explanation is apparent in the case of the other two men. They may have absorbed lead from some source in their food or they may have furnished false industrial histories. They both came from South Australia and it is possible that they may have been employed at the smelters, Port Pirie.

It will be seen that in general the lead excretion of these men is small and of the same order of magnitude as the lead excretion of normal individuals in Sydney.

*The Lead Excretion of Mine Workers.*

The mine workers in Table V include both surface workers and underground workers. As the conditions of exposure are not the same, the urinary excretion of the surface workers is shown separately in Table VII.

TABLE VII.

URINARY EXCRETION OF LEAD BY SURFACE WORKERS AT THE MINES.

| No. | Age. | Nationality. | Nature of Work. | Total Period of Work, Years. | Lead in Urine, Milligramme per Litre. |
|---|---|---|---|---|---|
| 28 | 19 | Australian | Labourer | 0·25 | 0·27 |
| 41 | 28 | Jugo-Slav | Labourer | 0·58 | 0·03 |
| 8 | 19 | Australian | Labourer | 1·0 | 0·09 |
| 37 | 22 | Australian | Labourer | 4·91 | 0·22 |
| 87 | 33 | Australian | Millhand | 13·5 | 0·02 |
| 44 | 22 | Australian | Labourer | 1·5 | 0·19 |

The number of subjects is too small for comparative purposes.

In the case of the underground workers examined the conditions under which these men work have already been stated. When an attempt is made to correlate the period of exposure with the lead excretion, many complications arise. In most cases the exposure has been intermittent, consisting of periods of mining work (surface and/or underground) alternated with bush or other non-mining work.

Table VIII shows the lead excretion of the underground workers whose exposure has been intermittent. The table is arranged to show the relation of the total period of exposure; that is the sum of all the periods of mining work to the lead excretion.

TABLE VIII.
THE LEAD EXCRETION OF UNDERGROUND WORKERS.
A. WITH INTERMITTENT EXPOSURE TO LEAD.

| Total Period of Mining Work. | No. | Age, Years. | Nationality. | Lead Excretion, Milligramme per Litre. |
|---|---|---|---|---|
| 1 year or less .. .. | 10 | 27 | Australian | <0·02 |
| | 21 | 22 | Australian | 0·08 |
| | 23 | 45 | Australian | 0·05 |
| | 25 | 21 | Australian | 0·11 |
| | 40 | 29 | Australian | <0·02 |
| | 42 | 38 | Italian | 0·09 |
| | 63 | 30 | Italian | 0·13 |
| | 71 | 32 | New Zealand | 0·17 |
| | 76 | 28 | Maltese | 0·09 |
| | 77 | 34 | Greek | 0·05 |
| | 103 | 42 | Italian | <0·02 |
| 1 to 5 years.. .. .. | 15 | 29 | Australian | 0·05 |
| | 18 | 49 | Australian | 0·02 |
| | 24 | 35 | English | 0·08 |
| | 26 | 30 | Bulgarian | 0·16 |
| | 29 | 26 | Maltese | <0·02 |
| | 30 | 34 | Maltese | <0·02 |
| | 31 | 34 | Maltese | 0·20 |
| | 38 | 38 | Italian | 0·11 |
| | 43 | 21 | South African | 0·29 |
| | 53 | 25 | Maltese | 0·06 |
| | 55 | 24 | Australian | 0·20 |
| | 56 | 41 | Maltese | 0·38 |
| | 57 | 46 | Indian | <0·02 |
| | 58 | 28 | Greek | 0·13 |
| | 59 | 33 | Italian | 0·05 |
| | 70 | 35 | Italian | <0·02 |
| | 73 | 41 | Italian | 0·02 |
| | 75 | 34 | Italian | 0·02 |
| | 79 | 29 | Greek | 0·01 |
| | 83 | 43 | Australian | <0·02 |
| | 84 | 22 | Australian | <0·02 |
| | 88 | 36 | Italian | 0·39 |
| | 89 | 30 | Jugo-Slav | 0·16 |
| | 92 | 30 | Jugo-Slav | 0·10 |
| | 95 | 31 | Italian | 0·44 |
| | 105 | 29 | Jugo-Slav | 0·42 |
| 6 to 10 years .. .. | 45 | 41 | Jugo-Slav | 0·19 |
| | 78 | 37 | Australian | 0·03 |
| | 80 | 35 | Australian | 0·18 |
| | 82 | 43 | Australian | 0·16 |
| | 85 | 31 | Maltese | 0·04 |
| | 86 | 35 | Australian | 0·02 |
| | 91 | 38 | Australian | 0·25 |
| | 100 | 43 | Australian | 0·22 |
| 11 to 20 years .. .. | 20 | 34 | Australian | <0·02 |
| | 22 | 35 | Australian | <0·02 |
| | 33 | 35 | Australian | <0·02 |
| | 54 | 43 | Australian | 0·20 |
| | 90 | 36 | Australian | 0·27 |
| | 93 | 39 | Australian | 0·20 |
| Over 20 years .. .. | 50 | 53 | Australian | 0·32 |
| | 109 | 52 | English | 0·17 |

It will be seen that the lead excretions cover a wide range, but there is a tendency towards low values, especially where the total period of work is under five years.

The lead excretion of those men in whom the exposure to lead has been continuous, that is the subject has worked underground continuously up to the time of examination, is shown in Table IX. In no case is there a continuous exposure greater than five years, on account of the general strike of 1922, when all the mines were closed down for a period of nineteen months.

From the table it can be seen that nearly all the subjects show high values, which are much greater than for persons not exposed to lead. High values were obtained with short as well as with long exposures.

TABLE IX.
THE LEAD EXCRETION OF UNDERGROUND WORKERS.
B. WITH CONTINUOUS EXPOSURE TO LEAD.

| No. | Continuous Exposure in Years. | Lead Excretion, Milligramme per Litre. |
|---|---|---|
| 68 .. .. .. | 0·08 | 0·15 |
| 52 .. .. .. | 0·33 | 0·37 |
| 51 .. .. .. | 0·58 | 0·18 |
| 69 .. .. .. | 0·58 | 0·13 |
| 74 .. .. .. | 0·66 | <0·02 |
| 67 .. .. .. | 0·75 | 0·49 |
| 94 .. .. .. | 0·91 | 0·29 |
| 106 .. .. .. | 0·91 | 0·47 |
| 62 .. .. .. | 1·0 | 0·11 |
| 107 .. .. .. | 1·0 | 0·19 |
| 39 .. .. .. | 1·5 | 0·17 |
| 108 .. .. .. | 1·66 | <0·02 |
| 110 .. .. .. | 1·66 | <0·02 |
| 64 .. .. .. | 1·83 | 0·14 |
| 81 .. .. .. | 2·0 | 0·11 |
| 65 .. .. .. | 2·25 | 0·19 |
| 88 .. .. .. | 2·5 | 0·39 |
| 60 .. .. .. | 2·66 | 0·36 |
| 96 .. .. .. | 2·83 | 0·25 |
| 97 .. .. .. | 3·0 | 0·15 |
| 98 .. .. .. | 3·0 | 0·13 |
| 66 .. .. .. | 4·91 | 0·28 |

*The Effect of a Period of Absence from Exposure on the Lead Excretion.*

Among the workers examined were many men who had previously worked at the mines, who had been absent for a period and were seeking reemployment at the time of examination. The effect of the period of absence on the urinary excretion of lead could therefore be studied. It was found that many men had had several periods of absence during their industrial life, thus complicating the study. There are, however, ten underground workers who have had a single exposure to lead of varying duration who have been away from the industry for a period and have now returned. The lead excretion of these men is shown in Table X.

It will be seen that the results after a period of absence from exposure show particularly low values in the amount of lead excreted. The first three miners had a continuous exposure to lead from two to three years, and after an absence from the industry for six to eight months their excretion of lead was found to be normal. The other members had shorter continuous exposures and longer absences with similar diminution of lead excretion.

**The Daily Excretion of Lead.**

The amount of lead excreted by underground workers per day in the urine and fæces was determined in a number of men who were in-patients of Broken Hill Hospital from causes unconnected with lead, such as fractures or herniæ. Where possible determinations were made on specimens collected from the same patient at intervals. All

TABLE X.
THE URINARY LEAD EXCRETION AFTER A PERIOD OF ABSENCE FROM EXPOSURE.

| No. | Work. | Period of Exposure, Years. | Period of Absence, Years. | Lead in Urine, Milligramme per Litre. |
|---|---|---|---|---|
| 29 | Miner | 2·33 | 0·5 | <0·02 |
| 15 | Miner | 3·5 | 0·66 | 0·05 |
| 30 | Miner | 3·16 | 0·66 | <0·02 |
| 53 | Trucker | 1·75 | 1·41 | 0·06 |
| 17 | Various | 2·58 | 1·5 | 0·08 |
| 40 | Miner | 0·41 | 1·58 | <0·02 |
| 42 | Mullocker | 1·0 | 2·66 | 0·09 |
| 4 | Miner | 1·33 | 3·0 | <0·02 |
| 10 | Mullocker | 1·0 | 12·0 | <0·02 |
| 23 | Miner | 0·5 | 15·0 | 0·05 |

specimens represented the excretion per twenty-four hours. The results are given in Table XI.

In Table XI the period of exposure has been continuous in subjects numbers 60, 70, 108 and 110. In the other cases the period of exposure is the sum of several exposures. It will be seen that the greater amount of lead is excreted in the fæces and that the bowel can excrete large amounts. There appears to be no relationship between the amount excreted in the urine to that in the fæces. It would appear also that the kidney has a limited power of excretion.

### Summary.

1. The lead excretion in the urine of one hundred men at Broken Hill has been quantitatively determined with the following results:

(i) The amount of lead excreted by men who had not been exposed to the dust of the mines, is in general very small and of similar magnitude to that of normal individuals in Sydney.

(ii) The amount of lead excreted by men exposed to the lead containing dust of the mines shows considerable variation in the different individuals examined. Those men who had been continuously exposed, were found to have high excretions of lead, which are much greater than for persons not exposed to lead. A high excretion of lead was found with short as well as with long exposures.

(iii) The lead excretions of different individuals after various periods of absence from exposure gave low values. Six months after exposure had ceased, individuals were found to be excreting normal amounts of lead.

(iv) Where the exposure was of an intermittent nature, the lead excreted by different individuals varied in amount, but with a tendency to low values, especially where the total period of mining work was short.

2. The total output of lead in the urine and fæces per day was determined with the following result:

(i) Lead is present in greater amount in the fæces than in urine in every man examined.

(ii) The excretory power of the kidney appears to be limited, while the bowel can excrete large amounts of lead.

(iii) There is no relationship between the amount of lead in the urine to that of the fæces.

## LEAD EXCRETION OF WORKERS AT THE SMELTERS, PORT PIRIE.

By R. W. TANNAHILL, B.Sc., A.A.C.I.,
*Sydney.*

### Smelting Operations.

The operations at the smelters are principally concerned with the production of refined lead from the ore concentrates received from the Broken Hill mines. The crude material is of four types.

1. Granular concentrates. The approximate composition is: lead 62%, sulphur 16%, zinc 9%, arsenic 0·2%, antimony 0·15%.

TABLE XI.
DAILY LEAD EXCRETION IN THE URINE AND FÆCES OF UNDERGROUND WORKERS.

| No. | Age. | Nationality. | Work. | Total Exposure, Years. | Date. | Lead. | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | Urine. | | Fæces. | | Total. |
| | | | | | | Total Milligrammes (Twenty-four Hours). | Milligramme per Litre. | Total Milligrammes. | Milligrammes per 100 Grammes. | Excretion, Milligrammes per Twenty-four Hours. |
| 50 | 53 | Australian | Miner | 25 | 4. 7.27 | — | — | 1·60 | 1·68 | — |
| | | | | | 11. 7.27 | — | — | 0·92 | 1·33 | — |
| | | | | | 19. 7.27 | 0·35 | 0·32 | 1·99 | 2·21 | 2·34 |
| 60 | 21 | Australian | Miner | 3 | | 0·47 | 0·36 | 1·02 | 1·24 | 1·49 |
| 70 | 35 | Italian | Miner | 4 | 10.10.27 | <0·02 | 0·01 | 3·86 | 12·87 | 3·88 |
| | | | | | 5. 1.28 | 0·08 | 0·05 | 1·67 | 9·28 | 1·75 |
| 71 | 32 | New Zealander | Miner | 0·75 | 10. 9.27 | 0·22 | 0·17 | 9·00 | 4·81 | 9·22 |
| | | | | | 26. 9.27 | 0·16 | 0·18 | 2·69 | 3·79 | 2·85 |
| | | | | | 3.10.27 | 0·15 | 0·10 | 3·49 | 3·32 | 3·64 |
| 72 | — | — | — | — | — | 0·14 | 0·18 | 0·19 | 0·21 | 0·33 |
| 101 | 40 | Australian | Miner | 5 | — | 0·44 | 0·37 | 1·42 | 1·29 | 1·86 |
| 108 | 32 | Italian | Miner | 2 | 2. 2.28 | 0·04 | 0·03 | 0·84 | 0·17 | 0·88 |
| | | | | | 1. 3.28 | 0·02 | 0·02 | — | — | — |
| 109 | 52 | English | Miner | 21 | 2. 2.28 | 0·17 | 0·11 | 0·23 | 0·46 | 0·40 |
| | | | | | 1. 3.28 | 0·04 | 0·02 | 0·47 | 0·25 | 0·51 |
| 110 | 30 | Swede | Miner | 2 | 2. 2.28 | 0·04 | 0·03 | 0·21 | 0·05 | 0·25 |
| | | | | | 1. 3.28 | 0·03 | 0·02 | 0·25 | 0·09 | 0·28 |

2. Slime concentrates. The approximate composition is: lead 56%, sulphur 17%, zinc 13%, arsenic 0·08%, antimony 0·15%.

3. Crude siliceous ore, containing about 20% lead.

4. Residues from the electrolytic zinc refining process, containing about 18% of lead.

The treatment of the ore concentrates consists of three distinct processes, roasting, smelting and refining.

*Roasting.*

The crude ore is roasted and sintered on coke-fired Dwight-Lloyd machines. By this means most of the sulphur is removed and escapes into the air as sulphur dioxide by passing up the stack with the flue gases. The residual ore is broken up and conveyed to the blast furnace.

*Smelting.*

The roasted material is smelted in the blast furnace with coke and fluxes. Crude metallic lead is thus formed and is removed from the furnace at intervals by siphoning and allowed to run into moulds. The bars so formed are conveyed to the refinery. The slag is run off, granulated and sent to the dump. The blast furnace gases are delivered to the bag-house, arranged with bags to collect the fume. The bags are mechanically shaken and the collected material returned to the blast furnace.

*Refining.*

Refining consists in removing the valuable impurities from the crude lead, especially silver and gold, by the Parkes process. The process consists in adding zinc to the molten lead, the zinc forming an alloy with the impurities. The valuable impurities are recovered from the alloy by distilling off the zinc. Copper and antimony are removed in special furnaces and the final product is refined lead.

Zinc concentrates, both granular and slime, are treated at the smelters. The crude material is roasted in Skinner or Barrier roasters. The furnace gases, principally sulphur dioxide, are delivered to the sulphuric acid plant, where after purification they are converted into sulphuric acid by the contact process. The roasted ore is shipped to Tasmania for electrolytic refining.

*Conditions of Exposure.*

Lead is brought to Port Pirie in the form of lead sulphide in the concentrates and slimes from Broken Hill. These materials especially slimes in the absence of moisture produce a very fine dust, containing a high concentration of lead in the form of sulphide. During the process of treatment which requires the use of high temperatures and blasts, the atmosphere may contain finely divided metallic lead. The entry of these high concentrations of lead into the body by way of the respiratory and alimentary tracts makes a high degree of toxicity possible.

Another factor to be considered is the presence in the atmosphere of such gases as carbon monoxide and sulphur dioxide. It was found by the Royal Commission on Plumbism, South Australia, 1925,[1] that carbon monoxide was prevalent in certain stages of the operations and was present in the blood of the workers, especially those employed at the bag-house and the blast furnace. Sulphur dioxide permeates the whole works. Thus many workers at the smelters are not only exposed to the action of lead, but to the gases, carbon monoxide and sulphur dioxide, making the conditions of exposure more complicated than at Broken Hill. The lead is more concentrated and occurs not only in the form of the sulphide, but also as the oxide and as metallic lead. Further, it is not known how far the presence of the gases affects the absorption of lead into the body.

*Subjects.*

The men forming the subjects of this investigation were workers in the various departments of the smelters "reporting sick" at the Port Pirie Hospital. All had symptoms suggestive of lead poisoning.

Two groups were selected: (i) Men with no previous exposure to lead who had been employed for less than one year; (ii) men who had worked five years or more.

Specimens of urine and fæces were collected from each individual. In the case of in-patients several specimens were obtained at intervals. Complete industrial histories were recorded.

*The Lead Excretion of Laboratory Workers at Port Pirie.*

The amount of lead excreted in the urine of workers in the Commonwealth Health Laboratory, Port Pirie, is shown in Table XIII.

TABLE XII.
LEAD EXCRETION IN URINES OF LABORATORY WORKERS, PORT PIRIE.

| Subject. | Lead in Urine, Milligramme per Litre. |
|---|---|
| K.C. | 0·12 |
| | 0·20 |
| C.S.M. | 0·23 |
| R.S. | 0·19 |
| | 0·20 |
| | 0·18 |
| | 0·12 |
| | 0·20 |
| | 0·18 |
| | 0·16 |
| | 0·20 |
| | 0·20 |
| | 0·19 |
| | 0·17 |

It will be noted that the amount of lead excreted by each individual is high and much greater than, for normal individuals in Sydney. A number of determinations on the same individual shows that the lead excretion keeps fairly constant. It is obvious that lead absorption is taking place, although the laboratory is situated three-quarters of a mile away from the smelters.

**Lead Excretion of Workers at the Smelters.**

*Men with Short Exposures.*

The amount of lead excreted in the urine and fæces in the case of men with exposures of less than one year is shown in Table XIII.

TABLE XIII.
LEAD EXCRETION IN URINE OF PORT PIRIE SMELTER EMPLOYEES WITH LESS THAN ONE YEAR'S EMPLOYMENT.

| No. | Age. | Nationality. | Exposure in Months. | Where Chiefly Employed. | Lead Excretion. | | |
|---|---|---|---|---|---|---|---|
| | | | | | Date. | Urine, Milligramme per Litre. | Fæces, Milligrammes per 100 Grammes. |
| 1001 | 23 | Australian | 6 | D. & L. | 8. 7.27<br>20. 7.27<br>29. 7.27<br>5. 8.27 | 0·27<br>0·11<br>0·07<br>0·02 | —<br>1·69<br>1·56<br>2·15 |
| 1002 | 23 | Australian | 4 | T.F. | — | 0·10 | 0·93 |
| 1003 | 24 | German | 7 | D. & L. | — | 0·14 | 4·08 |
| 1005 | 23 | South African | 7 | T.F. | — | 0·09 | — |
| 1007 | 23 | English | 5 | T.F. | 30. 7.27<br>3.10.27 | 0·30<br>0·52 | 2·84<br>3·37 |
| 1008 | 22 | English | 8 | T.F. | — | 0·07 | 1·16 |
| 1009 | 25 | Scotch | 7 | S.R. | — | 0·32 | — |
| 1010 | 24 | English | 3 | T.F. | — | 0·16 | — |
| 1011 | 30 | Australian | 6 | D. & L. | 11. 8.27 | 0·13<br>0·04 | 6·31<br>1·40 |
| 1013 | 23 | English | 5 | D. & L. | — | 0·22 | 1·65 |
| 1014 | 26 | Australian | 11 | D. & L. | 29. 9.27<br>10.10.27<br>17.10.27 | 1·04<br>0·63<br>0·32 | 4·93<br>3·13<br>— |
| 1015 | 42 | English | 7 | T.F. | 7.10.27<br>25.10.27 | 0·32<br>0·21 | 6·86<br>0·82 |
| 1017 | 24 | English | 8 | T.F. | 4.10.27<br>13.10.27<br>21.10.27 | 0·48<br>0·27<br>— | 2·82<br>3·28<br>0·55 |
| 1023 | 26 | German | 9 | Various | — | 0·17 | 0·15 |
| 1025 | 27 | Australian | 3 | T.F. | — | 0·62 | 2·18 |

D. & L. = Dwight-Lloyd roaster.
T.F. = Top floor of blast furnace.
S.R. = Skinner roaster.

The amount of lead excreted in the urine is variable, ranging from small amounts to very high amounts as in Number 1014. When several determinations were made on the same individual the amount of lead excreted became less when the subject was removed from exposure.

The lead in the fæces is also variable and the greater proportion of the lead absorbed is excreted in this way. No relationship appears to exist between the amount of lead in the urine to that of the fæces.

It will appear that the conditions at certain parts of the plant predispose to lead absorption, namely, the Dwight-Lloyd roaster and the top floor of the blast furnace.

*Men with Exposures Greater than Five Years.*

The lead excretion of these men is shown in Table XIV.

The lead output of this group varies although the men have been exposed to smelter conditions for a number of years.

TABLE XIV.
EXCRETION OF LEAD IN URINE OF PORT PIRIE SMELTER EMPLOYEES WITH MORE THAN FIVE YEARS' EMPLOYMENT.

| No. | Age. | Nationality. | Total Exposure in Years. | Last Period of Continuous Exposure in Years and Occupation. | Date. | Lead Excretion. | |
|---|---|---|---|---|---|---|---|
| | | | | | | Urine. | Fæces. |
| | | | | | | Milligramme per Litre. | Milligrammes per 100 Grammes. |
| 1004 | 36 | Greek | 7 | 5 on T.F. | — | 0·16 | 1·11 |
| 1006 | 39 | Australian | 18 | 18 as pipefitter | 13. 3.27<br>28. 9.27 | 0·06<br>0·07 | 2·54<br>3·77 |
| 1016 | 42 | Russian | 6·5 | 5 in refinery | — | 0·07 | 0·84 |
| 1021 | 44 | Australian | 24·5 | 17 in machine shop | — | 0·34<br>0·30 | 1·11<br>— |
| 1022 | 45 | German | 10 | 0·33 as painter | — | 0·30 | 0·12 |
| 1026 | 57 | Australian | 27 | 6 in refinery | 7.12.27<br>16.12.27 | 0·62<br>0·30 | 1·17<br>1·0 |

### Summary and Conclusions.

A quantitative study of the excretion of lead in the urine and fæces of workers at the smelters, Port Pirie, has been carried out, but the small number of subjects does not permit of too extensive an analysis of the results. It can be seen, however, that the main channel of excretion is by way of the fæces and that the urinary excretion ranges from normal amounts to as large a quantity as 1·04 milligrammes per litre (Number 1014).

That the urinary excretion is not related to the period of exposure is shown in Table XV.

TABLE XV.
RELATION OF THE PERIOD OF EXPOSURE TO THE URINARY LEAD EXCRETION.

| No. | Continuous Exposure in Months. | Lead in Urine, Milligrammes per Litre. |
|---|---|---|
| 1025 | 3 | 0·62 |
| 1010 | 3 | 0·16 |
| 1002 | 4 | 0·10 |
| 1007 | 5 | 0·30 |
| 1013 | 5 | 0·22 |
| 1001 | 6 | 0·27 |
| 1011 | 6 | 0·13 |
| 1003 | 7 | 0·14 |
| 1005 | 7 | 0·09 |
| 1009 | 7 | 0·32 |
| 1015 | 7 | 0·32 |
| 1008 | 8 | 0·07 |
| 1017 | 8 | 0·48 |
| 1023 | 9 | 0·17 |
| 1014 | 11 | 1·04 |

In general, it may be said that the same variability in the amount of lead excreted is found at Port Pirie as at Broken Hill. A comparison of similar groups is shown in Table XVI.

TABLE XVI.
MEN WITH LESS THAN ONE YEAR'S CONTINUOUS EXPOSURE TO LEAD.

| Period of Exposure, Months. | Port Pirie. Lead in Urine, Milligrammes per Litre. | Broken Hill. Lead in Urine, Milligramme per Litre. |
|---|---|---|
| 4 | 0·10 | 0·37 |
| 7 | 0·14 | 0·18 |
| | 0·09 | 0·13 |
| | 0·32 | — |
| | 0·32 | — |
| 9 | 0·17 | 0·49 |
| 11 | 1·04 | 0·29 |
| | — | 0·47 |

### Reference.

(1) Report of the Royal Commission on Plumbism, South Australia, 1925.

## THE BLOOD OF WORKERS EXPOSED TO LEAD.

By R. Y. MATHEW,
*Medical Officer in Charge, Commonwealth Health Laboratory, Port Pirie, South Australia.*

### General.

The following observations on the blood findings of workers exposed to lead are based upon the examination of the blood of over one thousand men who were working or had worked at the Broken Hill Associated Smelters Proprietary, Limited, Port Pirie, South Australia. The examinations were carried out at the request and for the information of the men's respective medical attendants when there was evidence of a varying degree suggestive of lead poisoning. The period covered by the examinations is from September, 1924, to February, 1928.

*Smelter Operations.*

A description of the operations at the smelters has been given under the article "Lead Excretion of Workers at the Smelters, Port Pirie."

In April, 1925, the amount being treated approximated 7,500 tons weekly, of which 5,000 tons were raw lead-bearing material, the balance being fuel and fluxes. The output of market lead was from 120,000 to 130,000 tons per year, which represented the largest individual production of market lead of all smelters in the world. In addition to the smelting operations about 1,250 tons of zinc concentrates (5·5% to 6% of lead) were desulphurized weekly and the calcines shipped to Risdon.

Numerous changes in plant and methods of treatment have been made in recent years with the object of eliminating risks and providing safeguards from risks of lead poisoning. Some of these alterations were made prior to the publication of the recommendations of the Royal Commission on Plumbism, 1925, and many have been made since then.

*Numbers of Employees and Cases of Lead Poisoning.*

Table XVII gives information regarding the total number of employees, average daily strength and number of cases of lead poisoning in half-yearly periods from 1924 to 1927.

TABLE XVII.
NUMBERS OF EMPLOYEES AND CASES OF LEAD POISONING, BROKEN HILL ASSOCIATED SMELTERS, PORT PIRIE, SOUTH AUSTRALIA.

| | June, 1924. | Dec., 1924. | June, 1925. | Dec., 1925. | June, 1926. | Dec., 1926. | June, 1927. | Dec., 1927. |
|---|---|---|---|---|---|---|---|---|
| Total number of individuals employed in half-year periods .. | 2,552 | 2,615 | 2,856 | 2,660 | 2,636 | 2,559 | 2,318 | 2,064 |
| Average daily strength .. | 1,595 | 1,601 | 1,698 | 1,750 | 1,673 | 1,691 | 1,641 | 1,565 |
| Total number of men suffering from lead poisoning in half-year periods .. | 103 | 131 | 130 | 48 | 60 | 38 | 29 | 54 |
| Men with lead poisoning as percentage of total number employed in half-year periods | 4·036 | 5·010 | 4·551 | 1·804 | 2·276 | 1·480 | 1·251 | 2·616 |

### Blood Examinations.

*Nature of Blood Examinations.*

Full clinical records of all persons whose blood was examined, were not available, but in the majority of cases there was either definite or suggestive evidence of lead poisoning. The examinations conducted throughout have embraced the estimation of hæmoglobin, the enumeration of red cells and an examination of the stain film. In the examinations of the blood films attention has been directed to the appearance of the red cells. In many cases enumeration and differential counts of white cells have been made. During 1925 these were carried out in one hundred consecutive specimens. The results did not give encouragement to

the view that these examinations would be of value as an aid to the diagnosis of lead poisoning and they were not continued as a routine part of the examination.

*Hæmoglobin Estimations.*

Estimations have been made by the Haldane method (507) and the Sahli method (510). The Sahli readings have been recorded as read on the scale 80 = normal and have not been converted to percentages. In regard to 140 men simultaneous estimations have been made by the Sahli method and by the method described by H. S. Newcomer,[1] the colorimeter reading being converted to grammes per hundred cubic centimetres by means of his table. The ranges of hæmoglobin found by the different methods are shown in Table XVIII in which

TABLE XVIII.

COMPARISON OF HÆMOGLOBIN VALUES AND ABSENCE OR PRESENCE OF PUNCTUATE BASOPHOLIA.

A.—*Films not showing Punctate Basophilia.*

| | Above 100% | 100–91% | 90–81% | 80–71% | 70–61% | 60–50% | Below 50% | Total. | Average. |
|---|---|---|---|---|---|---|---|---|---|
| (*a*) Determined by Haldane's Method .. .. | 6 | 26 | 24 | 14 | 3 | — | — | 73 | 88·2% |
| | Above 100 | 100–91 | 90–81 | 80–71 | 70–61 | 60–50 | Below 50 | Total. | Average. |
| (*b*) Determined by Sahli's Method (80=normal) | 4 | 34 | 80 | 54 | 11 | 3 | — | 186 | 83·9 |

| | Grammes per 100 Cubic Centimetres. | | | | | | | | | | Total. | Average. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Over 17 | 17–16·1 | 16–15·1 | 15–14·1 | 14–13·1 | 13–12·1 | 12–11·1 | 11–10·1 | 10–9 | Below 9 | | |
| (*c*) Determined by Newcomer's Method .. .. | — | 2 | 5 | 7 | 14 | 10 | 1 | — | — | — | 39 | 14 |

B.—*Films showing Punctate Basophilia.*

(*a*) Determined by Haldane's Method.

| Degree of Punctate Basophilia. | Percentage Hæmoglobin. | | | | | | | Total. | Average. |
|---|---|---|---|---|---|---|---|---|---|
| | Above 100 | 100–91 | 90–81 | 80–71 | 70–61 | 60–50 | Below 50 | | |
| Very slight .. .. .. | — | 7 | 19 | 14 | 2 | — | — | 42 | 83·3% |
| Slight .. .. .. | 1 | 5 | 18 | 13 | 2 | — | — | 39 | 80·8% |
| Moderate .. .. .. | 1 | 2 | 11 | 11 | 3 | 2 | — | 30 | 81·7% |
| Extensive .. .. .. | — | 1 | 6 | 10 | 4 | — | — | 21 | 78·5% |
| Very extensive .. .. | — | 1 | 4 | 4 | 1 | — | — | 10 | 81·3% |
| Extreme .. .. .. | — | — | — | — | — | — | — | — | — |
| TOTAL .. .. | 2 | 16 | 58 | 52 | 12 | 2 | — | 142 | — |

(*b*) Sahli's Method.

| Degree of Punctate Basophilia. | Above 100 | 100–91 | 90–81 | 80–71 | 70–61 | 60–50 | Below 50 | Total. | Average. |
|---|---|---|---|---|---|---|---|---|---|
| Very slight .. .. .. | 2 | 3 | 16 | 26 | 11 | 1 | — | 59 | 78·2 |
| Slight .. .. .. .. | 2 | 7 | 33 | 41 | 15 | 1 | — | 99 | 79·3 |
| Moderate .. .. .. | — | 2 | 7 | 24 | 14 | 6 | — | 53 | 74·2 |
| Extensive .. .. .. | — | 5 | 12 | 19 | 19 | 9 | — | 64 | 73·2 |
| Very extensive .. .. | — | 2 | 3 | 11 | 18 | 11 | — | 45 | 70·6 |
| Extreme .. .. .. | — | — | — | — | 1 | 3 | — | 4 | 58·0 |
| TOTAL .. .. | 4 | 19 | 71 | 121 | 78 | 31 | — | 324 | — |

(*c*) Newcomer's Method.

| Degree of Punctate Basophilia. | Grammes per 100 Cubic Centimetres. | | | | | | | | | | Total. | Average. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Above 17 | 17–16·1 | 16–15·1 | 15–14·1 | 14–13·1 | 13–12·1 | 12–11·1 | 11–10·1 | 10–9 | Below 9 | | |
| Very slight .. .. .. | — | — | 1 | 1 | 4 | 5 | 5 | 1 | 1 | — | 18 | 12·5 |
| Slight .. .. .. .. | — | — | — | 1 | 11 | 7 | 4 | 6 | — | — | 29 | 12·2 |
| Moderate .. .. .. | — | — | — | 1 | 2 | 3 | 5 | 3 | 2 | — | 16 | 11·5 |
| Extensive .. .. .. | — | — | — | 3 | 3 | 1 | 5 | 7 | 1 | 1 | 21 | 11·7 |
| Very extensive .. .. | — | — | 1 | — | 1 | — | 5 | 5 | 1 | 1 | 14 | 11·2 |
| Extreme .. .. .. | — | — | — | — | — | — | 2 | 1 | — | — | 3 | 11·2 |
| TOTAL .. .. | — | — | 2 | 6 | 21 | 16 | 26 | 23 | 5 | 2 | 101 | — |

TABLE XIX.

COMPARISON OF HÆMOGLOBIN VALUES AND RED BLOOD CELLS.

(*a*) Haldane.

| Hæmoglobin Value. | Red Blood Cells per Cubic Millimetre. | | | | | | | | | Total. | Average. |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | 6·6 million and over. | 6·5–6·1 | 6–5·5 | 5·5–5·1 | 5–4·6 | 4·5–4·1 | 4–3·6 | 3·5–3·1 | Less than 3·0 | | |
| Over 100% .. .. .. | — | 4 | 4 | — | — | — | — | — | — | 8 | 6·0 |
| 100–91% .. .. .. | — | 1 | 12 | 19 | 5 | 2 | — | — | — | 39 | 5·4 |
| 90–81% .. .. .. | — | — | 15 | 35 | 22 | 2 | 1 | — | — | 75 | 5·6 |
| 80–71% .. .. .. | — | — | — | 6 | 21 | 28 | 9 | 2 | — | 66 | 4·9 |
| 70–61% .. .. .. | — | — | 2 | 2 | 4 | 3 | 2 | — | — | 13 | 4·7 |
| 60–50% .. .. .. | — | — | — | — | — | — | 1 | 1 | — | 2 | 3·7 |
| Below 50% .. .. | — | — | — | — | — | — | — | — | — | — | — |
| TOTALS .. .. | — | 5 | 33 | 62 | 52 | 35 | 13 | 3 | — | 203 | — |

(*b*) Sahli. (80 = normal.)

| Hæmoglobin Value. | Red Blood Cells per Cubic Millimetre. | | | | | | | | | Total. | Average. |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | 6·6 million and over. | 6·5–6·1 | 6–5·5 | 5·5–5·1 | 5–4·6 | 4·5–4·1 | 4–3·6 | 3·5–3·1 | Less than 3·0 | | |
| Over 100 .. .. .. | 1 | 1 | 6 | 2 | 1 | — | — | — | — | 11 | 5·8 |
| 100–91 .. .. .. | 2 | 2 | 23 | 13 | 11 | — | — | — | — | 51 | 5·5 |
| 90–81 .. .. .. | 3 | 4 | 24 | 65 | 43 | 4 | 2 | 2 | — | 147 | 5·2 |
| 80–71 .. .. .. | — | 2 | 15 | 58 | 72 | 21 | 8 | — | — | 176 | 4·9 |
| 70–61 .. .. .. | — | — | 3 | 11 | 36 | 18 | 9 | 5 | — | 82 | 4·5 |
| 60–50 .. .. .. | — | — | — | — | 5 | 8 | 7 | 8 | 1 | 29 | 3·9 |
| TOTALS .. .. | 6 | 9 | 71 | 149 | 168 | 51 | 26 | 15 | 1 | 496 | — |

the groups are divided according to absence or presence of punctate basophilia in the blood film. Table XIX shows the ranges of hæmoglobin found in groups according to the red cell count.

It will be noted that most frequently the degree of punctate basophilia varies indirectly and the red cell count directly with the hæmoglobin value. There have, however, been numerous exceptions and wide differences have been observed in estimations done by the different methods. In simultaneous estimations by the Sahli and Newcomer methods, though mostly closely corresponding, divergent results have been observed, in some cases the former and in others the latter method being the

TABLE XX.

COMPARISON OF PUNCTATE BASOPHILIA AND RED CELLS IN 712 EXAMINATIONS FROM APRIL 27, 1925, TO MARCH 10, 1928.

(a) *Films not showing Punctate Basophilia.*

| | Red Blood Cells per Cubic Millimetre. | | | | | | | | | Total. | Average. |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | 6·6 million and over. | 6·5–6·1 | 6–5·6 | 5·5–5·1 | 5–4·6 | 4·5–4·1 | 4–3·6 | 3·5–3·1 | 3·0 and less. | | |
| Number .. .. .. | 5 | 8 | 62 | 107 | 64 | 9 | 2 | 1 | 1 | 259 | 5,340,000 |

(b) *Films showing Punctate Basophilia.*

| Degree of Punctate Basophilia. | Red Blood Cells per Cubic Millimetre. | | | | | | | | | Total. | Average. |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | 6·6 million and over. | 6·5–6·1 | 6–5·6 | 5·5–5·1 | 5–4·6 | 4·5–4·1 | 4–3·6 | 3·5–3·1 | 3·0 and less. | | |
| Very slight .. .. .. | 1 | 4 | 13 | 36 | 27 | 10 | 0 | 1 | — | 92 | 5,170,000 |
| Slight .. .. .. | — | 3 | 16 | 42 | 60 | 12 | 5 | 1 | — | 139 | 5,031,000 |
| Moderate .. .. .. | — | — | 6 | 19 | 31 | 8 | 8 | 3 | — | 75 | 4,809,000 |
| Extensive .. .. .. | — | — | 8 | 17 | 31 | 11 | 13 | 5 | — | 85 | 4,670,000 |
| Very extensive .. .. | — | — | 3 | 7 | 14 | 15 | 6 | 3 | — | 48 | 4,583,000 |
| Extreme .. .. .. | — | — | — | 1 | 1 | 1 | — | — | 1 | 4 | 4,200,000 |
| TOTALS .. .. | 1 | 7 | 46 | 122 | 164 | 57 | 32 | 13 | 1 | 443 | — |

TABLE XXI.

COMPARISON OF NATIONALITIES AND RED CELL COUNTS IN 286 EXAMINATIONS MADE FROM SEPTEMBER 1, 1924, TO APRIL 26, 1925.

(a) *Films not showing Punctate Basophilia.*

| Nationality. | Red Cell Counts. | | | | | Total. | Average Red Cell Counts. |
|---|---|---|---|---|---|---|---|
| | 6,000,000 and over. | 5,500,000 to 6,000,000. | 5,000,000 to 5,500,000. | 4,500,000 to 5,000,000. | 4,000,000 to 4,500,000. | | |
| British .. .. .. | 2 | 8 | 32 | 20 | 7 | 69 | 5,200,000 |
| Greeks .. .. .. | 4 | 18 | 38 | 20 | 6 | 86 | 5,500,000 |
| Maltese .. .. .. | — | 6 | 4 | 6 | — | 16 | 5,100,000 |
| Italian .. .. .. | — | 3 | 5 | 6 | 2 | 16 | 5,000,000 |
| Other Nationalities .. | 1 | 4 | 6 | 3 | 1 | 15 | 5,200,000 |
| | 7 | 39 | 85 | 55 | 16 | 202 | — |

(b) *Films showing Punctate Basophilia.*

| Nationality. | Red Cell Counts. | | | | | Total. | Average Red Cell Counts. |
|---|---|---|---|---|---|---|---|
| | 6,000,000 and over. | 5,500,000 to 6,000,000. | 5,000,000 to 5,500,000. | 4,500,000 to 5,000,000. | 4,000,000 to 4,500,000. | | |
| British .. .. .. | — | — | 7 | 9 | 6 | 22 | 4,700,000 |
| Greeks .. .. .. | 1 | 7 | 15 | 23 | 3 | 49 | 5,000,000 |
| Maltese .. .. .. | — | — | 1 | 3 | 2 | 6 | 4,700,000 |
| Italian .. .. .. | — | — | — | 1 | 1 | 2 | 4,500,000 |
| Other Nationalities .. | — | — | 1 | 3 | 1 | 5 | 4,600,000 |
| | 1 | 7 | 24 | 39 | 13 | 84 | — |

higher. In simultaneous estimations by the Sahli method wide differences have been observed when using two instruments made by different firms.

*Red Cell Count.*

The original Thoma and Thoma-Zeiss hæmocytometers were at first used for enumeration of red cells. For the last two years a double chambered apparatus of the Burker pattern with Neubauer ruling has been used and for the last month a Levy counting chamber with improved Neubauer single ruling and Trenner automatic pipettes, certified by the United States Bureau of Standards. Simultaneous counts made with all four instruments indicate that the Levy and Burker chambers are more easily manipulated and give less variable results than the Thoma and the Thoma-Zeiss and that the Levy gives a slightly lower count than the others. Counts ranging from 4·5 to 6·5 millions have been found both in healthy individuals who had not been exposed to lead, and in lead workers who had definite evidence of lead poisoning, and whose films showed extensive punctate basophilia, polychromasia and anisocytosis.

Many counts have been unexpectedly high; rarely have they been unexpectedly low. Little information of diagnostic value has been gained from red cell counts in the majority of cases and it seems probable that apart from errors of technique a large variation in the red cell count of an individual may be observed from day to day and perhaps during the day. An increase or decrease of as much as 500,000 has been observed from day to day in a normal individual whose initial count was 5,500,000. On the assumption that this was largely due to technical errors, similar counts have been repeated on other individuals, frequently revealing variations just as large. Table XXI shows comparisons between the extent of punctate basophilia and the red cell count.

*The Stained Film.*

The main and most constant changes that have been observed are anisocytosis, polychromasia and punctate basophilia of the red cells. Poikilocytosis has been observed less frequently and the presence of nucleated red cells in only a few cases. The presence of polychromasia and anisocytosis has been observed in the absence of punctate basophilia, but almost invariably accompanies punctate basophilia, the degree varying directly with the extensiveness of the punctate basophilia.

**Punctate Basophilia.**

*Methods of Staining for Demonstration of Punctate Basophilia.*

The following methods have at different times been used and have generally given satisfaction, some at times more than others: Leishman, Giemsa, Wright, Barlow, Unna, Michaelis, Manson, Glibert and Sellers.

Simple methylene blue stains are to be preferred to compound stains. For routine examinations the stains described by Sellers and Glibert are both recommended. Details of the methods of staining are shown in Appendix II. The frequency of punctate basophilia among the examinees is given in Tables XXII and XXIII.

TABLE XXII.

COMPARISON OF NATIONALITIES AND OCCURRENCE OF PUNCTATE BASOPHILIA IN 300 EXAMINATIONS MADE FROM SEPTEMBER 1, 1924, TO APRIL 26, 1925.

| Nationality. | Punctate Basophilia. | | | Changes other than Punctate Basophilia.[1] | No Abnormalities Observed. | Total. |
|---|---|---|---|---|---|---|
| | Slight. | Moderate. | Extreme. | | | |
| British .. | 12 | 7 | 5[2] | 22 | 46 | 92 |
| Greek .. | 33 | 10 | 7 | 28 | 65 | 143 |
| Maltese .. | 2 | 4 | — | 4 | 11 | 21 |
| Italian .. | 1 | 1 | — | 5 | 12 | 19 |
| Other Nationalities | 5 | 2 | — | 5 | 13 | 25 |
| | 53 | 24 | 12 | 64 | 147 | 300 |

[1] This includes polychromasia, anisocytosis, and poikilocytosis.
[2] In one case numerous normoblasts were observed.

TABLE XXIII.

OCCURRENCE OF PUNCTATE BASOPHILIA IN 715 EXAMINATIONS MADE FROM APRIL 27, 1925, TO MARCH 10, 1928.

| Punctate Basophilia. | Number of Films. | Percentage. |
|---|---|---|
| No changes .. .. .. .. | 259 | 36·2 |
| Very slight .. .. .. .. | 91 | |
| Slight .. .. .. .. .. | 138 | |
| Moderate .. .. .. .. .. | 83 | |
| Extensive .. .. .. .. .. | 85 | 63·8 |
| Very extensive .. .. .. .. | 55 | |
| Extreme .. .. .. .. .. | 4 | |
| TOTAL .. .. .. .. | 456 | |
| GRAND TOTAL .. .. | 715 | 100 |

In Table XXIII a much higher percentage of punctate basophilia is shown than in Table XXII. A greater percentage of those shown in Table XXIII was British. Some punctate basophilia may have been missed in the earlier series on account of poorly stained films.

*Relation of Extent of Punctate Basophilia to Period of Exposure.*

The method of indicating the degree of punctate basophilia present has been by the use of the terms slight, moderate, extensive *et cetera*. Actual counts have been made of the blood cells of about one hundred and fifty persons and hereunder is shown roughly how the terms would compare with the numbers estimated by a count. Even portions of the film were selected in which counts of all the red cells in a number of fields had averaged one hundred per field. The counts of the cells showing punctate basophilia were then based on a count of one hundred fields.

| | |
|---|---|
| Very slight .. .. | Up to 100 per million |
| Slight .. .. .. | 100 to 500 per million |
| Moderate .. .. | 500 to 2,500 per million |
| Extensive .. .. | 2,500 to 10,000 per million |
| Very extensive .. | 10,000 to 30,000 per million |
| Extreme .. .. .. | 30,000 upwards per million |

The shortest interval between the beginning of exposure and the examination of the blood has been three weeks. In this case there was definite evidence of lead poisoning and extensive basophilic degeneration. Several examinees had over thirty years' exposure. The majority of the examinations was made after an exposure period of from three months to five years. There have been instances of men with long exposure and men with short exposure showing extensive punctate basophilia of red cells and also little or none. All degrees of punctate basophilia have been found for different periods of exposure. It has not been practicable to examine the blood of men periodically from the beginning of exposure, a procedure which would be of value. In some cases punctate basophilia has largely disappeared in a few days after exposure has ceased and in others it has persisted for several months. In some cases it has practically disappeared at a second examination made a few days later and again been present at a third examination made a few weeks later. This has been noticed both in men with and in men without a further exposure. By a great many the period of exposure has been spent in a number of different jobs, frequently in different parts of the same departments, sometimes in different departments.

*Relation of Extent of Punctate Basophilia to Severity of Clinical Condition.*

The symptoms most frequently complained of are diminished working capacity, loss of appetite, loss of weight, unpleasant taste in mouth, abdominal pains, colic, constipation, pains in the limbs and joints, sleeplessness and headache. The signs most frequently observed are pallor, wasting, as evidenced by loss of subcutaneous fat, lead line on the gums, loss of muscle tone, colic, tenderness along peripheral nerves and anæmia. It has been found that the examination of the blood for punctate basophilia is of considerable value as a confirmatory sign in the case of men with fairly short exposure (up to three years) who complain of some or all of the above symptoms in a greater or less degree. When the symptoms complained of are severe and the signs are unmistakable, there is generally an extensive degree of punctate basophilia present. When the symptoms complained of are slight and the signs are little evident or absent, there is usually little more than a slight or moderate degree of punctate basophilia present.

*Relation of Punctate Basophilia to Nature of Work.*

Certain departments offer a greater exposure to dust and fumes than others and particular occupations in these departments apparently subject the worker to more prolonged or intense exposure than is undergone by other workers in the same department. Many of the men had during the period of exposure worked on a number of different jobs in the same or different departments. Punctate basophilia has been observed in the blood of men from practically every department and in the blood of men such as carpenters, fitters and stable hands, whose occupations may only occasionally require their working in close proximity to dust or fumes.

*Relation of Punctate Basophilia to Age and Nationality.*

Punctate basophilia has been observed in the blood of men of all ages from twenty to sixty-five and as shown in Table XXII in men of British and in men of foreign nationality.

*Types of Granules in Cells Showing Punctate Basophilia.*

Though a complete record has not been kept of the types of granules present in cells showing punctate basophilia, pronounced differences in size of the granules have been evident. A note has frequently been made when the predominant cell has been one containing very fine granules or when cells containing coarse or very coarse granules have been present. Though granules of all sizes from the very fine to the coarse have been observed and there can be no sharp differentiation between cells containing granules of different sizes, the cells that have been observed may be roughly divided into the following different classes:

Cells with very numerous fine granules.
Cells with less numerous medium granules.
Cells with still less numerous coarse granules.
Cells with mixed granules.

The mixed granular cells may contain any combination of two of the other three varieties of granules or all three. The finely granular cell has been the predominant cell in the blood of the majority of those with short exposure, mild symptoms and extensive punctate basophilia and the medium granular cell most frequently in cases with short exposure, more severe symptoms and extensive punctate basophilia. The latter also has been the predominant cell in those men with long exposure, severe symptoms and extensive punctate basophilia. The medium and coarse and mixed cells have been fairly uniformly evident in men with long exposure, mild symptoms and extensive punctate basophilia and both fine and medium granular cells in men with short exposure, slight symptoms and slight punctate basophilia.

The medium granular cells have been more evident in men with short exposure, severe symptoms and only slight punctate basophilia. The medium and coarse granules and mixed cells have been found in those with long exposure showing only slight punctate basophilia with either slight or severe symptoms, with perhaps a greater frequency of the medium cells in those with the more severe symptoms. When the coarse and mixed granular cells alone have been present, other changes in the red cells, such as anisocytosis and polychromasia, have been much less frequently observed than when there have been present cells of the fine and medium granular type, either alone or together. In about six men with short exposure, severe symptoms and extensive punctate basophilia the predominant cell has been a very coarsely granular one, the granules being much larger than those of the cells described above as coarsely granular. The cells have presented an appearance not unlike a speckled bird's egg, the granules being of irregular shape and intensely stained. On two occasions where re-examination was possible, these cells have been still present after an interval of one week, but not present after an interval of another week, though numerous granular cells of the other types were present.

Re-examinations have not been possible in the majority of those examined, but, when re-examinations have been made, marked changes in the extent of punctate basophilia have been observed, both in men without and in men with further exposure. In those without further exposure in whose blood the predominant cell has been the finely granular one, there is frequently little or no punctate basophilia evident at the end of a week or a month and when present the cells observed are more often of the medium granular type. In men without further exposure in whose blood the predominant cell has been the medium granular one, there is frequently a great reduction in the numbers of these cells present at the end of a month or two months. The punctate basophilia then present is frequently a mixture of medium, coarse and mixed cells. In men without further exposure in whose blood the predominant cells have been the coarse or mixed granular ones, and there is frequently not more than a slight or moderate degree of punctate basophilia present in these men, there is often little or no change in the extent or type of punctate basophilia present after a period of several months. In men with further exposure there is a tendency towards the development of larger granules and more granular cells than were previously present.

The kinds of granular red cells observed may be compared with those described in "A Report on the Value of Blood Examinations in the Control of Industrial Lead Poisoning" by Arthur Sellers, of the Department of Bacteriology and Preventive Medicine, Manchester University. Sellers describes three types of punctate red cells.

Type I is a red cell containing very numerous minute granules.

Type II contains less numerous and distinctly larger granules. The granules are generally scattered uniformly and are often more or less angular.

Type III contains large granules more rounded in outline and less numerous.

It is an uncertain and difficult matter to effect a classification of punctate red cells by microscopical characters. There are many intermediate types and there is no sharp dividing line between one type and another and it is by no means certain that the apparent size of the granule is not determined by the staining process. Moreover, many corpuscles are to be found containing minute granules along with larger ones. This routine system of classifying and counting kinds of punctate corpuscles was adopted with no immediate object in view, it was merely a convenient record of descriptions of microscopical appearances with the idea that something might eventuate by work of that nature. . . . . Type I cell is by far the most common and is sometimes the sole type present. It is the type of cell which usually appears first after exposure has commenced, also the first to vanish when exposure has ceased. Type II is usually present along with I or III or both. It frequently appears soon after exposure has commenced and remains present for long periods. Type III is usually accompanied by I and II, but it sometimes occurs alone. It is not present shortly after exposure to lead has commenced. It may persist for a long time. There is some indication of a relationship between microscopical characters of punctate red cells and the quantity of lead absorbed or the mode of absorption. It seems possible that the presence of finely granular cells is an indication of recent absorption and their presence over long periods of time suggests the intermittent absorption of fairly large doses. The presence of Types II and III on the other hand is rather indicative of chronicity or a more continuous absorption of smaller doses.

Though not particularly looked for the appearance and occurrence of cells showing punctate basophilia have been very similar to those described by Sellers with the exception that the medium and not the finely granular cell has been the one most frequently observed. This may be due in part to two different reasons: (i) Many cells which he would include in Type I, have been included in the medium granular type; (ii) in the presence of a predominance of medium granular cells many cells of the finely granular type may have escaped notice.

### BROKEN HILL AND PORT PIRIE INVESTIGATION.

#### The Examination of Blood Films Received from Broken Hill.

Six blood films were received from each man investigated at Broken Hill and were each stained by a different method, the combination of methods being varied in order to obtain a comparison of the results obtained by the different methods.

In the first few sets of films equal attention was given to all films, one hundred fields of each being examined. In the first fifty-six sets examined practically no punctate basophilia was observed.

After moderate or extensive punctate basophilia had been observed in all slides of several sets, the practice of staining and examining one film stained by Sellers's method first was introduced. If no punctate basophilia was observed, less attention was given to the other films. When punctate basophilia was present, either one hundred or fifty or twenty-five fields were examined in each of the other films; the total number of cells showing punctate basophilia, the number of fields examined and an estimate of the number of punctate cells per million red cells were recorded. In some cases a classification of the punctate red cells was made on the lines described by Sellers.

Some indication of the results found by the different staining methods is shown in Table XXIV, hereunder, which gives the actual counts.

TABLE XXIV.

COMPARISON OF PUNCTATE BASOPHILIA DEMONSTRATED BY DIFFERENT STAINING METHODS.

| Method. | Number of Fields Examined. | Cells showing Punctate Basophilia. | | | Total. | Per Million. |
|---|---|---|---|---|---|---|
| | | Type I. | Type II. | Type III. | | |
| Giemsa .. | 70 | 5 | 23 | 3 | 31 | 4,400 |
| Michaelis .. | 100 | 6 | 15 | — | 21 | 2,100 |
| Unna .. .. | 50 | 1 | 5 | — | 6 | 1,100 |
| Glibert.. .. | 70 | 1 | 7 | — | 8 | 1,100 |
| Leishman .. | 80 | 3 | 12 | — | 15 | 1,850 |
| Sellers .. .. | 80 | 14 | 17 | 3 | 34 | 4,200 |
| Glibert.. .. | 30 | 7 | 7 | 1 | 15 | 5,000 |
| Michaelis .. | 30 | 4 | 8 | 12 | 12 | 4,000 |
| Leishman .. | 30 | 4 | 8 | — | 12 | 4,000 |
| Wright.. .. | 30 | 3 | 7 | — | 10 | 3,300 |

The types of cells showing punctate basophilia were similar to those found in lead workers at Port Pirie, though the presence of an extensive degree of punctate basophilia was much less frequent. A record was kept of the presence of polychromasia, anisocytosis and poikilocytosis.

In the earlier films received a differential count of white cells was made, but it was not carried out in the later ones.

#### Port Pirie Examinees.

The Port Pirie examinees were selected for the most part from men having the necessary periods of exposure who were sent to the laboratory for blood examinations, and all to some degree complained of symptoms suggestive of lead poisoning. Many had highly suggestive signs and several were definitely suffering from lead poisoning. It was not possible in all cases to obtain the first specimen of urine and fæces and the second and third samples were rarely obtained. The industrial histories showed that many of the men had worked on several jobs. The small numbers of men in each group do not warrant statistical tables or deductions.

In some cases the blood examinations were apparently confirmatory of and in others contradictory to other evidence. The significance of the amounts of lead in the urine does not appear to be constant. Amounts from 0·05 to 0·20 milligramme of lead per litre were repeatedly found in the urine of the members of the laboratory staff who are not exposed to lead except in the forms of dust from the wharf, half a mile away and fumes from the smelter three-quarters of a mile away. The amounts found in duplicate samples sent to Sydney for check estimations closely correspond with these findings.

The largest amount found in the series was in No. 1014, an initial estimate of 1·04 milligrammes per litre being followed by ones of 0·63 and 0·32 milligramme per litre at successive estimations during the succeeding three weeks. Though complaining of symptoms suggestive of lead poisoning, his physical condition and active participation in a strenuous sport during this period were incompatible with the symptoms complained of. With the exception of this man the presence of more than 0·3 milligramme per litre has in all cases been accompanied by definite evidence of lead poisoning. All examinees with definite evidence of lead poisoning have not, however, shown so great an amount of lead in the urine.

#### *The Occurrence of Punctate Basophilia.*

At Broken Hill twelve out of one hundred and seven workers examined showed punctate basophilia, but in four of the examinees the basophilia did not exceed 500 cells per million. In Port Pirie of the twenty-one examinees punctate basophilia was demonstrated in eighteen. Tables XXV and XXVI furnish further details regarding those presenting punctate basophilia. It will be seen from these tables that no relationship between the occurrence of basophilia and the amount of lead excreted in the urine is apparent.

TABLE XXV.
BROKEN HILL CASES SHOWING BASOPHILIC DEGENERATION (OVER 200 CELLS PER MILLION).

| No. | Punctate Red Cells, per Million. | | Urinary Excretion, Milligramme per Litre. | Total Exposure, in Years. |
|---|---|---|---|---|
| | Minimum. | Maximum. | | |
| 63 | 200 | 300 | 0·13 | 0·5 |
| 88 | 200 | 300 | 0·39 | 2·5 |
| 90 | 200 | 300 | 0·27 | 12·58 |
| 84 | 300 | 500 | 0·02 | 2·41 |
| 81 | 500 | 800 | 0·11 | 2·0 |
| 109 | 500 | 600 | 0·11 | 21·25 |
| 59 | 600 | 800 | 0·05 | 1·41 |
| 64 | 600 | 3,000 | 0·14 | 1·83 |
| 66 | 800 | 3,000 | 0·28 | 4·91 |
| 61 | 1,300 | 1,700 | — | 22·0 |
| 58 | 2,000 | 4,000 | 0·13 | 2·83 |
| 86 | 3,000 | 5,000 | 0·02 | 10·25 |

Nos. 63, 88, 90 and 84 are doubtful cases.

TABLE XXVI.
PORT PIRIE CASES SHOWING BASOPHILIA (OVER 200 CELLS PER MILLION)

| No. | Punctate Red Cells, per Million. | Urine, Milligrammes per Litre. | Group. |
|---|---|---|---|
| 1003 | 500 | 0·14 | Men with no previous exposure and with less than one year in the industry. |
| 1014 | 300 | 1·04 | |
| | | 0·63 | |
| | | 0·32 | |
| 1001 | 600 | 0·07 | |
| | 700 | 0·11 | |
| 1008 | 800 | 0·07 | |
| 1009 | 1,000 | 0·32 | |
| 1007 | 2,000 | 0·30 | |
| 1005 | 2,000 | 0·09 | |
| 1010 | 3,000 | 0·16 | |
| 1001 | 4,000 | 0·27 | |
| 1011 | 6,000 | 0·13 | |
| 1017 | 8,000 | 0·48 | |
| 1002 | 10,000 | 0·10 | |
| 1015 | 12,000 | 0·32 | |
| | | 0·21 | |
| 1025 | 30,000 | 0·62 | |
| 1002 | 200 | 0·30 | Men exposed for more than five years. |
| 1006 | 500 | 0·06 | |
| 1004 | 1,600 | 0·16 | |
| 1021 | 3,000 | 0·34 | |
| 1026 | 3,000 | 0·30 | |

### Summary.

The observations made and inferences drawn in the foregoing paragraphs are, no doubt, extremely vague. Full and exact personal histories were not available in the majority of examinees nor was re-examination often possible. Though the exact interpretation of the blood examination of a worker exposed to lead is not always definite, the advisability of having the results of a blood examination available in a lead worker suspected to be suffering from lead poisoning for consideration with other evidence of lead poisoning is quite apparent.

The blood picture may undergo rapid and extraordinarily great changes.

A single examination, therefore, is not sufficient and in addition to examinations of the blood after the beginning of illness, periodical examinations from the time of entering the industry are highly desirable.

Most constant and characteristic changes are found in the red cells in the form of anisocytosis, polychromasia and punctate basophilia which can readily be ascertained from examination of the stained film.

The presence of punctate basophilia is a definite indication of the absorption of lead and the extent, type and degree of permanency of the punctate basophilia present give some indication as to the extent and degree of permanency of the bodily damage.

## APPENDIX II.

### METHODS OF STAINING BLOOD FILMS TO DEMONSTRATE PUNCTATE BASOPHILIA.

#### Compound Stains.

Leishman's, Giemsa's and Wright's methods have on occasions given good results; at other times they have been disappointing and unreliable and are not recommended. Simple stains have been found much more satisfactory than compound stains.

#### Simple Stains.

Harlow's method (modified), in which immersion of the slide in a solution of eosin is followed by immersion in a solution of methylene blue, has not been found to give much satisfaction. This method is described in an excellent publication on lead poisoning edited by R. M. Hutton and issued in 1923 by the provincial Board of Health of Ontario, whose Division of Industrial Hygiene uses the method and finds it exceedingly satisfactory.

Unna's and Manson's methylene blue methods have not been found as satisfactory as the methods described by Sellers and Glibert which are both being used as routine stains.

Details of these methods are as follows:

*Sellers's Method.*

To prepare the stain the following formula is used:

Methylene blue (pure medical): 1 gramme.
Sodium bicarbonate: 6 grammes.
Distilled water: 200 cubic centimetres.

The stain is good for six weeks.

To stain film: (i) fix with methyl alcohol for three minutes, (ii) stain for half a minute, (iii) wash well in tap water till the film becomes a pale greenish colour. Dry with blotting paper.

Appearance of cells showing punctate basophilia is as follows. The red cells are stained a faint greenish colour. The basophilic granules show as dark blue dots in the red cells.

*Glibert's Method.*

To prepare the stain use the following formula:

Methylene blue: 2 grammes.
Sodium bicarbonate: 12 grammes.
Distilled water: 200 cubic centimetres.

To stain the film: (i) fix in methyl alcohol for three minutes, (ii) stain for one minute (iii) wash well in distilled water until the colour almost fades. Dry with blotting paper.

The appearance of punctate cells is similar to that described for Sellers's method.

#### Schwarz's Thick Drop Method.

The thick drop method of Schwarz,[4] abstracted in the *Journal of Industrial Hygiene*, has not been found to give much satisfaction. The stain used is Manson's borax methylene blue and it is said that an examination of ten fields gives as much information as the examination of two hundred fields in an ordinary smear preparation.

## THE DISTRIBUTION OF LEAD IN THE BODY AFTER ABSORPTION.

By R. W. TANNAHILL, B.Sc., A.A.C.I.,
*Sydney.*

In order to determine the fate of the lead after absorption a chemical examination was made of material obtained *post mortem* from two workers who had been exposed to lead, one at Broken Hill and the other at Port Pirie.

The material, which consisted of the whole or portions of viscera, was dispatched to Sydney, preserved in 50% spirit. On arrival the organs were removed from the preservative, roughly dried with a towel and weighed. A portion was then taken for analysis and weighed and the lead determined by the chromate method as previously described.

The results of the analyses and related information of the two cases are as follows:

CASE I.—History: A male, aged 48, died of chronic nephritis and acute fibrinous pericarditis. He had been a miner for twenty-four years at Broken Hill and was working up to six weeks before death.

### Result of Analysis of Post Mortem Material.

The results obtained from the analyses of the *post mortem* material are given in Table XXVII.

TABLE XXVII.
CASE 1.—ANALYSES OF POST MORTEM MATERIAL.

| Material. | Wet Weight in Grammes. | Wet Weight of Sample for Analysis in Grammes. | Lead Found. | | |
|---|---|---|---|---|---|
| | | | Milligrammes in Sample. | Milligrammes per 100 Grammes. | Milligrammes in Whole Organ. |
| Lung .. | 740 | 172 | 1·89 | 1·1 | 8·1 |
| Liver .. | 1,058 | 162 | 1·75 | 1·07 | 11·32 |
| Heart .. | 450 | 167 | 0·47 | 0·28 | 1·26 |
| Spleen .. | 105 | 56 | 0·18 | 0·32 | 0·34 |
| Kidney .. | 56 | 26 | 0·10 | 0·39 | 0·21 |
| Muscle .. | 197 | 107 | 2·04 | 1·91 | — |
| Bone .. | 20 | 10 | 6·52 | 65·2 | — |
| Intestine | 12 | 12 | 0·78 | 6·5 | — |

*Comments.*

Column 4 of Table XXVII shows that the greatest concentration of lead is in bone, with much smaller amounts in intestine, muscle, lung, liver, kidney, spleen and heart in descending order. Considering the body as a whole it is evident that, although the actual amount cannot be calculated, as with other organs in column 5, the skeleton contains the bulk of the lead, while appreciable amounts are present in liver and lung.

As the chief portal of entry of the lead is by way of the respiratory tract, the concentrating of lead in the lung is only to be expected.

The large amount of lead in bone indicates that the lead is stored in the skeleton after absorption, while the presence of lead in the liver suggests that this organ is concerned in the excretory process by which lead is removed from the body in the fæces. The high concentration of lead in the portion of intestine examined (which was not washed out prior to analysis) adds confirmation to this fact.

*Solubility of the Lead Compound in Spirit.*

After the *post mortem* material had been left in contact with 50% spirit for a period of six weeks, the spirit was examined for lead and found to contain 9·6 milligrammes. As the spirit had been previously examined for lead and found to contain only a trace, this large amount must have come from either the container or the organs. An experiment showed that a similar quantity of spirit left in contact with the container for the same period of time contained 0·03 milligramme lead. Hence the lead was principally derived from the organs. These facts indicate that:

1. The lead in the tissues is easily extracted by alcohol. This should help in elucidating the manner in which lead is combined, and suggests an organic form of combination.

2. The inadvisability of dispatching material in spirit for analysis for lead, unless the analysis is carried out as soon as possible. In Case II which follows, this was done and the preserving fluid examined for lead at the same time as the organs.

CASE II.—History: A male, aged 52, died one day after operation for strangulated inguinal hernia. The total length of service was unknown, but he had been employed at the smelters, Port Pirie, for at least three years prior to death. He had been away with lead poisoning for two months and resumed work two days before operation.

### Result of Analysis of Post Mortem Material.

The results of analysis of the *post mortem* material are summarized in Table XXVIII.

TABLE XXVIII.
CASE 2.—ANALYSES OF POST MORTEM MATERIAL.

| Material. | Wet Weight in Grammes. | Wet Weight of Sample for Analysis in Grammes. | Lead Found. | | |
|---|---|---|---|---|---|
| | | | Milligrammes in Sample. | Milligrammes per 100 Grammes. | Milligrammes in Whole Organ. |
| Heart .. | 395 | 100 | 0·25 | 0·25 | 0·98 |
| Liver .. | 1,081 | 100 | 5·03 | 5·03 | 54·37 |
| Lung .. | 415 | 150 | 1·27 | 0·84 | 3·48 |
| Kidney .. | 115 | 100 | 4·50 | 4·50 | 5·17 |
| Muscle .. | 242 | 100 | 1·94 | 1·94 | — |
| Bone .. | 247 | 100 | 3·25 | 3·25 | — |
| *Débris* and spirit | — | — | 2·68 | — | — |

*Comments.*

In this case the concentration of lead is greatest in the liver with lesser concentrations in kidney and bone and small concentrations in lung and heart. Although the concentration in bone is not as high as in Case I, the skeleton is the chief storehouse for lead when the body is considered as a whole, while large amounts are present in liver, kidney and lung.

### Summary.

By a correlation of the various facts elicited by this investigation some idea of the mechanism of the absorption, distribution and storage of lead can be gained. It would appear that when lead is absorbed by way of the respiratory tract, it enters the systemic circulation and is carried to all parts of the body. A proportion of the lead reaches the

liver and is excreted with the bile into the intestine and removed from the body by the fæces. The kidney also removes lead from the blood, but its powers of excretion are limited and the lead in the urine never reaches the high values obtained in the fæces. The greatest amount of lead is taken up by the bones, where it enters into combination and is stored out of harm's way. Thus the large amount of lead in Case I indicates absorption of lead over a long period (*vide* history of case).

Other workers[6] have shown that under certain conditions, such as a diminished resistance, the store of lead in the bone may be released and give rise to a severe intoxication, with the result that the organs of excretion, the liver and kidney contain large amounts of lead. The results in Case II appear to be consistent with the explanation that a release of stored lead followed a condition of shock.

### References.


(1) L. T. Fairhall: "A Rapid Method of Analyzing Urine for Lead," Lead Studies XI, *Journal of Biological Chemistry*, 1924, Volume LX, page 485.

(2) C. Badham and H. B. Taylor: "Lead Poisoning—Concerning the Standards which Should be Used in Diagnosing this Industrial Disease, together with a New Method for the Determination of Lead in Urine," Studies in Industrial Hygiene, Number 7, contained in Report of the Director-General of Public Health, New South Wales, for the year 1925, page 52.

(3) D. Avery, A. J. Hemingway and V. G. Anderson: "Determination of Minute Amounts of Lead in Water, with Notes on Certain Causes of Error," *Proceedings of the Australian Institute of Mining and Metallurgy*, 1921, Number 43.

(4) M. Siegfried and W. Pozzi: "*Über die Bestimmung kleiner Bleimengen*," *Biochemische Zeitschrift*, 1914, *Band* LXI, *Seite* 149.

(5) J. C. Thresh: "The Estimation of Lead in Water and Urine," *The Analyst*, 1924, Volume XLIX, page 124.

(6) J. C. Aub, L. T. Fairhall *et alia*: "Lead Poisoning," *Medicine Monographs*, 1925, Volume IV, page 1.

(7) W. Denis and A. S. Minot: "A Method for Determination of Minute Amounts of Lead in Urine, Feces and Tissues," *Journal of Biological Chemistry*, 1919, Volume XXXVIII, page 449.

(8) A. S. Minot: "Lead Studies. II. A Critical Note on the Electrolytic Determination of Lead in Biological Material," *Journal of Biological Chemistry*, 1923, Volume LV, page 1.

(9) O. Schumm: "*Über den Nachweis kleiner Mengen von Blei im Harn*," *Zeitschrift für Physiologische Chemie*, 1922, *Band* CXVIII, *Seite* 189.

(10) G. Meillère: "*Recherche et Dosage Électrolytique du Plomb*," *Journal de Pharmacie et Chimie*, 1902, *Tome* XVI, *page* 465.

(11) G. Meillère: "*Isolement des Substances Minérales Contenues à l'État de Traces dans un Complexe Salin*," *Journal de Pharmacie et Chimie*, 1907, *Tome* XXVI, *page* 443.

(12) G. Meillère: "*Recherche Toxicologique du Plomb et Cas Particulier de l'Urine des Saturnins*," *Journal de Pharmacie et Chimie*, 1914, *Tome* X, *page* 225.

(13) V. Froboese: "*Beitrag zur Bestimmung von Blei in Organischen Substanzen besonders in Kot und Harn*," *Archiv für Hygiene*, 1925-1926, *Band* XCVI, *Seite* 289.

(14) C. K. Reiman and A. S. Minot: "A Method for Manganese Quantitation in Biological Material together with Data on the Manganese Content of Human Blood and Tissues," *Journal of Biological Chemistry*, 1920, Volume XLII, page 329.

(15) M. G. Mellon and H. F. Reinhard: "The Simultaneous Deposition of Lead and Lead Peroxide," *Proceedings of the Indiana Academy of Science*, 1921, page 181.

(16) H. B. Taylor: "The Determination of Lead in Urine," *Journal of the Royal Society of New South Wales*, 1927, Volume LXI, page 315.

(17) V. N. Ivanov: *Chemische Zeitschrift*, December, 1914.

(18) K. H. Mertens: "*Über den Einfluss des Chlorcalciums auf die Fällung des Bleis als Sulfid*," *Zeitschrift für Analytische Chemie*, 1895, *Band* XXXIV, *Seite* 453.

(19) L. Dede and P. Bonin: "The Action of Neutral Chlorides in Preventing Precipitation by Hydrogen Sulphide," *Chemical Abstracts*, 1922.

(20) W. B. S. Bishop: "The Occurrence of Lead in the Egg of the Domestic Hen," THE MEDICAL JOURNAL OF AUSTRALIA, April 21, 1928, page 480.


## Reviews.

### A BOOK ON DIET.

"PRACTICAL DIETETICS IN HEALTH AND DISEASE," by Dr. Sandford Blum, of San Francisco, is a book of three hundred and eighty pages containing dietaries "successfully employed" by the author during his twenty years of practice.[1]

It is divided into two parts, namely for adults and children, in both of which are found dietaries for those in health and disease.

Underneath the headings of diseases arranged alphabetically, are set out the foodstuffs recommended by the author. The quantitative method is not followed as fully as modern knowledge would warrant, even in the section on diabetes. The latter is rather unsatisfactory as may be inferred from the statement "while its inaccuracies are generally admitted, the caloric method may sometimes be advantageously employed."

In the section on infant feeding eight feedings are recommended at two months old and nothing but milk and water in varying proportions is advised for artificial feeding up to eleven months.

There is little mention of the principles of dietetics, as perhaps is to be expected in a book with the above title. On the other hand, there is a dogmatism which no doubt is hard to avoid in a book of this character. In the section dealing with normal diet adult males are told to avoid among other things, tongue, sausage, liver, stews, pork, duck, goose, herring, lobster, crab, radish, corn, cabbage, onion, cucumber, sprouts, turnips, berries, jam, nuts, pastry and cakes.

The author apparently belongs to the generation which was excessively occupied with the digestibility of foods; this is also suggested by the statement in the introduction that coarse foods cause an irritation in the intestines which in many cases is injurious.

Despite these adverse criticisms there is a great deal of information to be got from this well planned book.

### THE PUBLIC HEALTH IN TROPICAL COUNTRIES.

IN spite of the sacrifice of the indefinite article from the title "Manual of Hygiene and Public Health,"[2] Jahar Lal Das wins the eulogy of his sponsor, Lieutenant-Colonel W. C. Ross, I.M.S., that this text book for medical and public health students which "should also be of great use to politicians and the press, and to all who desire knowledge or require a handy book of reference," is "well

[1] "Practical Dietetics for Adults and Children in Health and Disease," by Sandford Blum, A.B., M.S., M.D.; Third Revised and Enlarged Edition; 1928. Philadelphia: F. A. Davis Company. Royal 8vo., pp. 390. Price: $4.00 net.

[2] "Manual of Hygiene and Public Health: A Text Book for Medical and Public Health Students," by Jahar Lal Das, D.P.H., with an introduction by Lieut.-Colonel W. C. Ross, M.B., Ch.B., D.P.H., I.M.S.; Second Edition; 1928. Calcutta: Butterworth and Company (India) Limited. Crown 8vo., pp. 661, with illustrations. Price: Rs. 3/8/- net.

written in a simple and clear manner, without the waste of words which is a sure sign of the want of knowledge."

As is the rule with such text books compiled in India, the author apparently takes all preventive medicine, hygiene and sanitation as his province. There are the useful chapters on air, ventilation, water, industrial hygiene, food, disposal of sewage and refuse, climate, meteorology, communicable diseases and vital statistics. These subjects are dealt with in the ordinary routine manner of the English text book with special adaptation to Indian conditions.

In addition, there is an excellent chapter on soil, which breaks comparatively new ground from the text book point of view with regard to microorganisms in soil, varieties of soil, made soil, dampness of soil, effects of irrigation on soil and diseases due to soil.

The chapter on house sanitation deals with construction of houses and is especially useful for sanitarians living in a tropical country.

The schedule of data required for a public health survey is excellent and embodies in the main those items which American text books and journals in their discursive surveys have made a special feature.

Probably the best chapter in the book is that dealing with medical entomology, now so large a feature of tropical hygiene.

Communicable diseases, with special emphasis on those prevalent in tropical and subtropical countries, are also a feature.

The final chapters on village sanitation, mass education, sanitation of fairs and festivals and camp sanitation should be of special value to those health officials, either governmental or municipal, in tropical countries with an indigenous and only partially educated population.

The book can be commended to any medical officer, health inspector, medical missionary or anyone who has to do with sanitation in eastern countries, and also must be of interest to all who specialise in preventive medicine.

## ULTRA-VIOLET RAYS AND HAIR LOSS.

DR. A. LORAND in his work, "The Ultra-Violet Rays," lays particular stress on the action of ultra-violet light on internal and nervous diseases and in preventing loss of colour and falling of the hair.[1] Quoting himself as an example of establishing hair growth on a bald head, he lays stress on the relation of the endocrine glands to obesity, old age, neurasthenia, Graves's disease, impotency and high blood pressure, as well as the falling and loss of colour of hair. The stimulation of the endocrine glands by the passage through them of the blood affected while in the skin by the ultra-violet rays is the source from which improvement springs. In cases of impotency the local application of rays is of great value in producing the desired result, care being exercised not to give an overdose to the sensitive skin. The subsequent application of red rays will prevent any skin trouble. The causes of hair loss are summed up as a deficient circulation of blood in the scalp due to physical causes, for example, tight hats or pressure during sleep, arteriosclerotic changes in the scalp which have been found to occur even in quite young people, alterations in the endocrine glands, especially the thyreoid and sex glands; occasionally nervousness would appear to be a cause also. Nervous disease and intestinal autointoxication are similarly blamed as well as obesity, gout and diabetes. To combat the hair loss it is necessary to remove the cause or causes outlined and to stimulate the growth of hair by improving the nourishment of the follicles. This is the part played by ultra-violet light.

[1] "The Ultra-Violet Rays," by Arnold Lorand, M.D.; 1928. Philadelphia: F. A. Davis Company. Royal 8vo., pp. 258. Price: $2.50 net.

The book is written in a semi-scientific strain. Much of the matter might well be omitted and the remainder considerably condensed. Its discursiveness is a decided drawback from the practical point of view. The author gives no clear indication of the exact technique employed, even of the particulars of the mode of treatment apart from this. While it may stimulate an interest in its readers regarding the possibility of curing or alleviating hair loss, it is difficult to understand to whom the book can make an appeal.

## A BOOK ON TONSILS AND ADENOIDS.

THREE or four years ago it was stated by a contributor to one of the journals devoted to the speciality of diseases of the ear, nose and throat, that he had discovered no less than two thousand articles written in one year and including correspondence in medical journals, on the subject of the tonsils. In "The Tonsils and Adenoids and Their Diseases," by Dr. Irwin Moore, lists of references numbering approximately six hundred are given.[1] These two facts indicate both the necessity for such a work as that at present under review and also the meticulous care and consideration that have been given to its compilation. Irwin Moore takes his reader back to very early days, mentioning the writings of Celsus, A.D. 10, who described the operation of "complete removal of the tonsils." In the very interesting pages devoted to historical features he points out that the early recognized difficulties and dangers of the operation are those still present today. The basis of the work, as pointed out in the preface, is largely the author's personal experience and knowledge and the name of Irwin Moore is a sufficient guarantee as to the solidity of that foundation. The lengthy and varied lists of references indicate that the final structure has not been completed without the reinforcement of the world-wide experience and wisdom of other practitioners of high standing. The foreword, written by John W. H. Eyre, indicates that the theme running through the book is focal sepsis. In the preface the author expresses the hope that "this monograph may be a guide to the busy practitioner and a handy reference to the specialist" and this is followed by a well compiled index, covering the nine chapters and indicating that the tonsils and adenoids are viewed from every standpoint. Anatomical conditions are naturally dealt with first and after bacteriology and pathological conditions, including systemic infections and general effects of the focal sepsis, have been considered, the author discusses fully the surgical removal of the tonsils. Irwin Moore can rest assured that the hope expressed in his preface is more than realized.

The subject of local anæsthesia may perhaps have received a little more generous treatment and strange to say under general anæsthesia no reference is made to intratracheal insufflation anæsthesia whereby blood and other noxious materials are effectively prevented from entering the lower air passages. In discussing complete removal by dissection he states: "A deep anæsthesia by means of open ether is indicated, so that both swallowing and cough reflexes are abolished. The importance of a skilled anæsthetist cannot be overestimated."

Various surgical procedures are portrayed in very great detail, but therein is not one useless word. Hæmorrhage and other complications are dealt with at great length. The book consists of some 359 pages, excluding the 35 pages devoted to an excellent analytical index. It is delightfully written, full of detail and well arranged, printed in good type and freely illustrated; it is a book that every general practitioner may read with profit and pleasure and one that may equally be recommended to the specialist.

[1] "The Tonsils and Adenoids and their Diseases: Including the Part they Play in Systemic Diseases," by Irwin Moore, M.B., C.M. (Edinburgh); 1928. William Heinemann (Medical Books) Limited. Demy 8vo., pp. 414, with illustrations. Price: 21s. net.

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## The Aerial Medical Service.

In the second half of 1927 the Australian Inland Mission evolved a scheme for the establishment of an aerial medical service in the sparsely populated portions of western and northern Queensland and Northern Territory where hardy men and women are living and working out of reach of medical aid. In our issue of November 12, 1927, we published full details of this scheme. In that article it was pointed out that an experiment would be made for the purpose of giving medical attendance to those needing it who would otherwise be unable to secure the services of a doctor, and of providing medical practitioners living and practising beyond the reach of a colleague with a competent practitioner when required for consultation or for assistance in the event of a surgical operation. It was further proposed that the flying doctor should be prepared to transport a patient when necessary by aeroplane to the nearest hospital. In March, 1928, we announced that Dr. K. St. Vincent Welch had undertaken to act as medical officer to the Aerial Medical Service for one year. It will further be remembered that the medical officer would not accept any fees and would not conduct private practice. He would receive his remuneration from the Inland Mission. The actual working of the scheme was to be controlled by a local committee acting under the direction of the Australian Inland Mission. Arrangements were made with an organization called the Queensland and Northern Territory Aerial Service, usually known as Qantas, to supply an aeroplane and a pilot to be stationed at Cloncurry. It was anticipated that the flying doctor would cover at least twenty thousand miles by air during the first year.

The first year's experiment is now drawing to a close and much experience has been gained. Dr. St. Vincent Welch has been at the beck and call of the people in an immense area extending as far north in Queensland as Vanrook, some seventy miles to the north of Normanton, as far north-west as Newcastle Waters, as far west as Avon Downs and Sedan, as far south as Bedourie. It has to be remembered that this district and the district adjoining to the south have been in the throes of a terrible drought for six or seven years. In many places the land is parched and burnt; the cattle have died by the thousand and the small farmers have been ruined. Under the best conditions the townships and the stations have contained a small population, small in relation to the enormous stretches of land. This population in several parts has dwindled and is at present a fraction of what it was. When the drought breaks, as it has already broken in several of these stricken areas, the people will return and a spell of prosperity may be anticipated. In some districts the flying doctor has been summoned for trivial injuries and illnesses; under other circumstances the hardy citizens who have learned to face trials and difficulties without demur, leave the call until it is very late. The movements of the flying doctor are known to every one and when a flight to some distant station or township is undertaken, people with minor ailments come in to see the doctor and thus they derive material benefit from the accident of a visit on account of serious illness or dangerous injury. Dr. St. Vincent Welch was given an enthusiastic reception on his arrival in Cloncurry and the places within the Barkly tableland. His organization has been eminently successful and the experiment has been fully justified. Sometimes he has travelled to a station or settlement by motor car, either because of the unsatisfactory conditions for flying or because it was thought that the interests of the patient would be better served. Sometimes the patient has been taken back by aeroplane; sometimes urgent treatment has been carried out at the homestead. It is true that there are times when the flying doctor has little or nothing to do for several days. But there are times when he is kept busy, giving medical aid either by himself or with

one of the medical practitioners in the far "outback." On the whole the months spent in organizing and starting the Aerial Medical Service have been full of incident and of achievement. A single life saved would have made it worth while; there have been several. He has given the people living in isolation in an immense tract of land confidence and a feeling of security and that is a most valuable thing. Dr. St. Vincent Welch will have the satisfaction on his return to New South Wales of knowing that he has sacrificed a year for the good of those courageous men and women who have learned to smile at Nature in her unkindest moods, and who endure without complaint years of drought and of seemingly unprofitable toil, waiting patiently for the rains that change the face of the earth in a single night. He will know that he has proved that even the most distant settlement can be brought within the reach of skilled medical aid. There is not only need for the continuation of the experiment on the Barkly tableland; sooner or later the same expedient must be adopted for the whole of the sparsely settled interior of this great continent. It is a matter of national importance that this scheme should be extended. But for the present we wish to appeal to the members of the medical profession to sustain the service. A practitioner with sound experience and resource is being sought to give up a year, as Dr. St. Vincent Welch has done. He should be self-reliant, courageous and athletic. He should have a keen sense of humour, for there is no more satisfactory antidote to tragedy. He must be prepared to tolerate extreme heat and relative cold on winter nights. He must be strong enough to work for work's sake and be satisfied with a small reward. If any payment is made for his services by the people in receipt of them, the money goes toward the expenses of the service. This position will be tempting to anyone in search of adventure and doubly so to a patriot who loves Australia and yearns to perform a national service. Those who are prepared to take up this work, are requested to communicate with the General Secretary, Australian Inland Mission, 92b, Pitt Street, Sydney, or with the Secretary of the Mission in Brisbane, Melbourne, Adelaide or Perth.

# Current Comment.

## SUSCEPTIBILITY TO INFECTION.

IT is usually assumed that resistance to bacterial infection is brought about by the action of immune bodies that either inhibit the multiplication of the bacteria or destroy the bacteria or that neutralize the products of the bacteria or that increase the power of the leucocytes to ingest the bacteria and to hold them prisoners until other processes lead to their death or removal. There is so much evidence in support of this hypothesis that it would be foolish to challenge it at the present time. It is further taught that infection follows when resistance is lowered, when the infecting dose of bacteria overwhelms the tissues locally or generally or when mechanical or chemical injury offers to the invading bacteria an opportunity of establishing themselves in a limited focus from which they can escape in a constant stream until a true infection of the body takes place. It is, however, by no means easy to define in words the physiology of susceptibility or to explain the essential differences between a susceptible and a resistant individual. It is reasonable to postulate that a further process is involved in the production of the state of tissue susceptibility than the permanent or temporary failure of the bactericidal, antitoxic or phagocytic activity of the tissue fluids. In dealing with this subject there is a grave danger of playing with words, of substituting fresh terms for undefined conceptions.

During the past few years W. F. Petersen has endeavoured to measure the constitutional reactivity of given individuals by determining the permeability of the capillary cells of the skin and the degree of inflammatory reactivity. The former is estimated by comparing the protein content of blister fluid with that of serum, while the latter is expressed by the quotient of the permeability rate divided by the blistering time. Petersen has determined both the permeability rate and the inflammatory index of persons in health and of those affected with a large variety of pathological conditions. For the purpose of our present discussion two observations are of especial importance. The first is that there is an unmistakable relationship between the height of the blood pressure and the permeability rate. The second is that in the stage following certain infections, such as influenza and scarlet fever, the permeability is increased. Other investigators have called attention to the fact that the sympathetic nervous system exercises an influence on metabolism and the extent of the tissue reaction.

E. F. Müller and W. F. Petersen have recently attempted to find an explanation for the behaviour of the tissues in connexion with infection.[1] They point out that the great majority of the tissue cells are not exposed to external influences, including bacteria. The skin and mucous membranes, on the other hand, can be attacked by bacteria from

[1] *Münchener Medizinische Wochenschrift*, December 14, 1928.

without and often carry on them even virulent pathogenic organisms. As long as the tissue protection or resistance is normal, no infection follows. As soon as certain events take place, such as a wound, a metabolic change or another form of stimulation, the local resistance is at once interfered with and an infection follows. From the local infection the bacteria seek to invade the body, but are encountered by the protective powers of the internal cells. Again something happens and the microorganisms overcome the opposition and a general infection results. The authors regard the protective mechanism of the tissue cells as a vital characteristic and the direct expression of living energy. The component cells of the body are subjected to the alternating inhibitory and stimulating sympathetic influences of the body as a whole. The inhibitory influences lead to a diminution of the circulation of the part and thus to a local contraction of the capillaries. They regard this diminished blood supply as the direct cause of tissue susceptibility. They adduce a considerable amount of experimental and other evidence to show that under certain circumstances changes of temperature give rise to a peripheral contraction of small vessels, both in the skin and mucous membranes and also in the mucous membranes of the intestines. Blood collects in the abdominal vessels and this increases the peripheral anæmia. The affected cells become increasingly permeable and in consequence lose their contained fluid; they decrease in size. The authors use a word that appears to us to be unfortunate, for it is impossible to define it. They state that these influences lead to an inhibition of the vitality of the tissue cells. If by vitality they mean tissue cells deprived of their power of absorbing food elements and of eliminating dissociation products of tissue changes, it would have been less ambiguous had they substituted the term anabolic power. Be this as it may, they assume that the continuance of the sympathetic impulse results in a removal of the barrier against the invasion of the body by the bacteria which have already gained an entrance into the surface tissue cells. They maintain that every sudden, intense increase of the functional activity of an abdominal organ is associated with a considerable local dilatation of blood vessels and an augmentation of the secretion of the organ. In connexion with this vascular change a reaction takes place associated with a diminution of the activity of the affected cells. When the body temperature is raised there is always an increased flow of blood through the abdominal organs resulting from a dilatation of the splanchnics. Similarly in the premenstrual period, during an attack of migraine, in gastritis and in certain hypersecretions of the stomach there is an abnormally large amount of blood driven through the dilated abdominal blood vessels. While this is going on, the peripheral vessels are contracted and the permeability of the peripheral tissue cells is diminished. They instance the appearance of a rigor in an acute infection as a manifestation of a disturbance of the sympathetic control of the vascular system. The skin fails to perform its normal functions in these conditions.

It will be recognized that no satisfactory explanation of "catching cold" has yet been abduced. The suggestion that chilling interferes with the normal bactericidal and phagocytic mechanism is unsatisfactory, since it leaves the main effect untouched. Müller and Petersen point out that exposure to cold, even if extreme, is neither harmful nor of ætiological significance, provided that the peripheral circulation and the circulation through the oral and nasal mucous membranes are maintained. A man can work in severe cold and keep quite well if his skin is well supplied with blood. He may even sweat. But if he stands in a cold environment and his skin becomes blue and bloodless, the sympathetic impulse will cause the abdominal vessels to dilate. The effect will be a lowered permeability of the cells of the oral and nasal mucosa, a loss of fluid from these cells and a reduction of their power of resisting bacteria.

The hypothesis that infecting bacteria penetrate the surface only when some external stimulus induces a sympathetic reaction, with a flooding of the abdominal organs with blood and a corresponding depletion of the peripheral vessels of blood appears to be rational and not unacceptable, but it does not satisfy every infective process. It is difficult to reconcile the onset of scarlatina or morbilli with the production of contraction of the tonsillar vessels. An explanation must be provided for the reaction of children who are susceptible to diphtheria to the Schick test. It would seem to be unlikely that the skin and oral and nasal mucous membranes of a susceptible child would be permanently of lowered "vitality," of lowered permeability. Rather would it be expected that the difference between the susceptible and the immune child as far as diphtheria is concerned, is a difference of immune body content of the child's body fluids. Clinical experience teaches that no external influence is needed for a susceptible child to become infected with diphtheria bacilli or with scarlatinal streptococci, when exposed to the infection. Again it would be very difficult to explain the great rarity of infection in an insusceptible child, since it cannot be rare for such a child to be subjected to some impulse on its sympathetic system at the same time as it is exposed to infection. The possibility that the sympathetic nervous system is unusually sensitive in persons who are susceptible to acute infections, cannot be excluded. But this would merely provide an explanation for a physiological change that could take place in the surface cells. We are therefore driven to the conclusion that the part played by an abnormal action of the sympathetic system in determining the susceptibility of the surface cells toward an invading pathogenic bacterium is not the only factor that determines the majority of acute infections. It certainly appears to be a sufficient explanation for the action of exposure to chilling in precipitating a coryza or an attack of pneumonia.

# Abstracts from Current Medical Literature.

## BACTERIOLOGY AND IMMUNOLOGY.

### Precipitation of Phosphate Crystals in Media.

SARA A. SCUDDER (*Journal of Bacteriology*, September, 1928) noted in 1924 minute crystals in veal infusion ascitic agar upon which gonococci were growing. The crystals had the microscopic morphology of the triple phosphates commonly found in alkaline urine. Similar crystals were found beneath the layer of growth in tubes of semi-solid hormone agar containing stock cultures of gonococcus, meningococcus and *Micrococcus catarrhalis*. Since the original observations records have been kept of the occurrence of these crystals and the influence of various media and procedures upon their formation has been studied in sixty-five strains of bacteria. The media used are described and it was found that bacteria vary in their power to form ammonium magnesium phosphate crystals in nitrogenous media. Favourable factors for crystal production are the presence of meat infusion, peptone and inorganic phosphate. Sodium chloride and moisture are inhibitory. The formation of these crystals is not considered of any importance at the present time, but the author considers that under properly controlled conditions with synthetic media of known composition the rate of crystal production may constitute a measure of bacterial utilization of amino acids with liberation of ammonia.

### Bacillus Proteus Infections.

JOHN F. TAYLOR (*Journal of Pathology and Bacteriology*, October, 1928) has examined fifty-three strains of *Bacillus proteus* recovered from human sources. All the strains studied were obtained in the ordinary routine investigation of cases and no selective methods were employed to discover *Bacillus proteus* in preference to other organisms. Each case has been considered on its history and clinical course as well as its bacteriological findings, before deciding whether *Bacillus proteus* had a pathogenic action or not. The pathological conditions are all classified and particulars are given. This worker has restricted the name *Bacillus proteus* to a well defined group of organisms. They are non-sporing, Gram-negative, pleomorphic bacilli which produce a spreading or creeping growth on solid media. They are proteolytic and hæmolytic. They do not ferment lactose, mannite or dulcite, but ferment dextrose and saccharose and occasionally maltose. True indol may or may not be formed from peptone water. In milk a transient clot is formed which is very rapidly peptonized. In the fifty-three strains recovered three fermented only maltose and these same three strains alone produced true indol. Agglutination tests showed variations between strains, but in no case did a serum fail to agglutinate one or more strains in addition to its homologous strain, though one serum almost attained this distinction. There is no differentiation as regards agglutination reactions between the sources of the organisms or their clinical pathogenicity. Absorption tests seem to show definite differences between strains. There was no tendency for strains to fall into groups whether they were derived from urine, fæces, pus or other sources or whether they were clinically innocent or virulent and no classification into pathogenic or non-pathogenic strains could be made by the laboratory methods employed. This work shows that *Bacillus proteus* may produce severe infection in the human subject or may exist as a harmless saprophyte in the tissue, body fluids or excreta. An attempt has been made to classify the strains as pathogenic or non-pathogenic on the history, clinical course and bacteriological findings in each case. *Bacillus proteus X* 19 of Weil and Felix was found to be serologically distinct from the fifty-three strains of *Bacillus proteus* collected, but otherwise to resemble them closely.

### Gram-negative Cocci of the Naso-Pharynx.

G. S. WILSON AND MURIEL M. SMITH (*Journal of Pathology and Bacteriology*, October, 1928) have examined seventy-eight strains of Gram-negative cocci, other than meningococci, obtained from the naso-pharynx of a number of healthy volunteers. The fermentation reactions were tested on a litmus-ascitic-agar medium containing 1% of glucose, maltose, mannite, lactose, sucrose or salicin and also in a 5% serum-peptone-water medium containing Anrade's indicator and 1% of the same sugars. The sugars were made up in a 10% solution in distilled water sterilized separately by steaming and subsequently added to the medium. The sugars were incubated for a fortnight, the results being read daily. In only one half of the fifty strains examined in both media were the results in agreement. The cause of this was not clear, but the fact has been noted by previous workers. The authors consider that fermentation tests do not afford a reliable means of distinguishing between different members of the group of Gram-negative cocci. The seventy-eight strains examined when grown on ascitic agar plates yielded after forty-eight hours' growth two types of colonies, the rough and the smooth, but after five days many of the primarily smooth colonies were transformed into the rough type. Experiments have shown that one and the same strain may be dissociated into smooth and rough variants and that smooth variants may be recovered from a pure culture of a rough variant. The growth in serum broth after twenty-four hours is subject to great variation in appearance; but on the whole the permanently smooth colonial types give rise to a powdery or finely granular deposit and the primarily and secondarily rough types to a coarsely granular deposit often accompanied by a surface ring growth. These workers conclude that the cultural and bio-chemical characters of the Gram-negative cocci are subject to such variation that they cannot justifiably be used for purposes of classification in the way in which they have previously been employed. They suggest that instead of dividing them up into a number of so-called species, *catarrhalis*, *flavus*, *cinereus*, *mucosus*, *siccus*, they should be grouped under the broad term *Neisseria pharyngis*, the characteristics of which are given in full.

### Hodgkin's Disease and Birds.

ÉLISE S. L'ESPERANCE (*Journal of Immunology*, March, 1928) has inoculated five healthy chickens intravenously with emulsions of lymphatic glands from two patients suffering from Hodgkin's disease. All the chickens developed either a typical or an atypical tuberculosis and in the tissue smears, stained by Ziehl-Neelsen method, acid-fast granules and rods, extracellular and intracellular, were demonstrated in two of them and non-acid-fast granules in one. Reinoculation of material from the lesions of one of these chickens into another chicken produced an identical though more extensive manifestation of the disease. Atypical tuberculosis developed in a guinea-pig by inoculation of the tissue from the fourth chicken. From this guinea-pig a pure growth of bacteria with the staining and cultural characteristics of the avian tubercle bacillus was produced on egg media. The author feels justified in concluding that the results obtained indicate that the ætiological agent in certain forms of Hodgkin's disease is pathogenic for birds or that the avian tubercle bacillus is a factor in producing some of the lesions which are interpreted as Hodgkin's granuloma.

### Enumeration of Bacteria in Milk.

W. R. ALBUS (*Journal of Bacteriology*, October, 1928) has brought forward evidence to show that the experimental methods employed to obtain the data upon which plate counts of the bacteria in milk have been so severely criticized, permit errors that may be responsible for the widely discrepant results. He has performed experiments to show that when a sample of milk in which bacteria are actively growing, is packed on ice for two hours, a number of the organisms are killed and also that the time consumed in the plating process will affect the accuracy of the plate counts if the organisms are actively multiplying, especially in milk of low bacterial content. The technique that should be used, is

described in detail and the author emphasizes the importance of making only one delivery from a single pipette, as the material clings to the inside of the pipette when a second delivery is attempted. The data given in this article prove that the plate count method when conscientiously performed by trained personnel, can and does give consistent counts when all sources of error are fully controlled.

## HYGIENE.

### Water in Swimming Baths.

G. K. BOWES (*The Journal of State Medicine*, September, 1928) describes the two methods in actual use in Birmingham for maintaining the water of swimming baths in a clean and wholesome condition. The first method used in the older baths consists in emptying and refilling the baths as often as may be needed. In actual practice this is usually found necessary every alternate day. After emptying and before refilling the baths are cleaned with "Chloros." In the more modern baths the water with which the bath is filled at the beginning of the season, is kept pure by continuous filtration. The water is drawn from the deep end of the bath by means of a pump, strained through metal gauze and after being passed through the pumps, is delivered under pressure on to the surface of the filters. Before filtration it receives an addition of alumina and soda ash to act as a coagulant. The filters are two in number and each consists of a cylindrical metal casing containing a thickness of 82·5 centimetres of sand resting on gravel. After passage through the filters the water is heated by passage through a steam heater, aerated and delivered to the shallow end of the bath. The maximum speed at which the filters are capable of working is 61,000 litres per hour. The filtration plant is in operation for one hour longer than the advertised time during which the bath is used. During the two consecutive summers 1925 to 1926 investigations were undertaken to determine the relative efficiency of these methods judged by the bacterial content of the water examined. In all five baths were examined, one with a capacity of 279,000 litres being taken as an example of a bath where emptying and refilling were used and four with modern filtration plants, one having a capacity of 430,000 and the other three 495,000 litres each. Samples were taken from each bath at intervals of about a week or more. A two days' gelatine count was taken and the number of bacteria capable of growth on agar at 37° C. was determined after two days. The presence or absence of *Bacillus coli* in measured quantities was determined by the presumptive test of production of acid and gas after forty-eight hours' incubation in McConkey's lactose-bile-salt medium. The chlorine content of the water was estimated in two series of samples taken from a bath equipped with a modern filtration plant. An increased amount of chlorine was found to be present. This increase was not very considerable and was probably due in part at least to urinary contamination. The results of the investigation are summarized as follows. Both in baths with filtration plant and in those depending on refilling for maintaining the water fresh, bacterial counts of the water both on gelatine and agar were generally much lower in colder than in warmer weather. In hot weather the water in baths with filtration plant was purer bacteriologically than the water in non-filtration baths on the second day after refilling. In cold weather this superiority was less. No immediate effect either of pollution added by bathers within a preceding period of a few hours nor of filtration during a similar period could be detected in a swimming bath specially examined from this point of view. In the great majority of samples examined the bacterial counts exceeded the most stringent standard adopted, while some of the more lax standards were also exceeded. In order to minimize the risk of transmission of disease it is probably desirable to adopt a bacterial standard for bathing water similar to that used for drinking water. It is possible to keep the water up to a drinking water standard or even a standard below this by the use of a disinfecting agent continuously present in the water, chlorine being the most obvious agent in a residual strength of from 0·2 to 0·5 part per million.

### A New Larvicide for Mosquitoes.

ROBERT MATHESON and E. H. HINMAN (*The American Journal of Hygiene*, March, 1928) while working on mosquito ecology endeavoured to determine the effect of high pH on larval development. In order to secure a pH above 8 they employed boric acid and potassium chloride as a buffer against sodium hydroxide. In standard solutions of pH 8·0, 8·6, 9·2 and 9·8 the lethal effect was so pronounced that they concluded that the death of the larvæ was not due to high hydrogen-ion concentration alone. They assumed that the action might be due to the presence of boron. It is well known that boron in excess is very injurious to plants and borax is used in destroying the larvæ of the common house fly. In order to settle this point two experiments were conducted with the same buffer solution, but with water instead of with sodium hydroxide. The pH of these solutions ranged from 6·7 to 7·0 during the course of the experiments. In each experiment two hundred *Culex pipiens* larvæ in various stages of development were used. In both instances all the larvæ died within three or four days. As a result of these experiments it was decided to test as a larvicide some compound containing boron. Commercial borax was selected for this purpose. From these experiments it was found that borax in concentrations of 1·5 or more grammes per litre of water proved an efficient larvicide for mosquito larvæ. Such concentrations of borax retained their lethal action for a considerable period of time. It is believed that borax will prove a practical and safe larvicide in cisterns, rain-water barrels and in any type of container where water is stored for purposes other than that of drinking. The borax would need to be renewed, as such containers are emptied of their contents and refilled by fresh water. It may also prove effective in various types of temporary and permanent pools where the plant and animal life are of little importance. In such places it may be necessary to renew the borax supply only at long intervals. This must be determined by further experimental work. It should be possible to manufacture a borax of sufficient purity for use as a larvicide at a very low cost.

### Death from Electric Shock.

THE ENGINEERING COMMITTEE OF THE CONFERENCE ON ELECTRIC SHOCK (*The Journal of Industrial Hygiene*, April, 1928) has issued a report with the purpose of presenting the statistics of electric fatalities in the United States and Canada and of comparing the conditions there with those of Switzerland and of England and Wales. The rates of both the United States and Switzerland are decidedly higher than those of England and Wales and the rates of the United States registration area are slightly and significantly higher than those of Switzerland. The disparity between the figures for the United States and those for England and Wales is explicable in part by the fact that there is a much greater use of electricity *per capita* in the United States; consequently there are more high tension generating stations and in terms of percentage of total population more persons exposed to hazards from electricity. Yet this explanation alone is not sufficient to account for the difference in the figures. As to the disparity between the Swiss and the English figures, the Swiss conditions are probably more like those in the United States and the Swiss accident death rate from electricity is but little better than that of the United States. The Committee recommends that attempts to reduce electric fatalities should be adopted by education of the general public in precautionary measures and in the importance of using only first-class electrical household equipment. Any sort of equipment can now be plugged into electrical outlets, although the actual wiring is somewhat controlled by building, fire and insurance regulations. Continued education of the employees of the electrical industries themselves by the methods already in force and development of methods of resuscitation for persons with cardiac failure as well as for those with purely respiratory failure, should also be adopted.

# Correspondence.

## OBSTETRICAL PRACTICE.

SIR: I am sorry that Dr. Hubert Jacobs should have mistaken my description of his writing for personal abuse; I have never met the gentleman and had no reason to abuse him.

Frank comment is commonly regarded as discourteous when uncomplimentary; that charge, therefore, worries me not at all.

Dr. Jacob's intemperate language in comment on my work is understandable in view of his mistake; he was smarting from a fancied insult, but the bad taste exhibited by his silly bombastic signature is neither understandable nor excusable.

If *accouchement forcé* ever was an obstetric fashion or if ever there was an attempt to make it so, it was over fifty years ago and must have deservedly fallen rapidly into disrepute.

Dr. Jacobs appears to think that I am unaware that I am at variance with certain authorities whom he names. He might more briefly have written all obstetrical authority and I should have agreed with him, for I know of none who agrees with me.

I am not unacquainted with the analyses of cases treated in maternity hospitals. Dr. Jacobs has fallen into the common error of reading them with an uncritical mind. Had he not done so, it would not be necessary for me to now point out to him that their operative cases, including the forceps deliveries, are all of them cases complicated in some way or other or cases maltreated by some incompetent general practitioner before they reached the hospital. Naturally such cases, as I have already pointed out, yield a higher rate of mortality and morbidity than the uneventful and uncomplicated cases. The mortalities and morbidities were due to the antecedent trouble, not to the operative procedures or forceps that were resorted to to relieve them.

The accepted standards of obstetrical practice are not founded on any scientific basis, but result from the frightful case mortality that followed all operative interference in the days when our forefathers attended their accouchment cases in their old soiled frock coats.

I have satisfied myself that the simple operations of manual dilatation and forceps delivery are not fraught with danger, unless they be unduly delayed when indicated. I find that in recent years I have manually dilated in about 24% of my cases and I have had neither mortalities nor morbidities; what I have done, others may do.

Dr. Jacobs mistakes the simplicity of my "routine" for crudity. In my hands this routine has successfully stood the test of one thousand cases and it is adapted to the circumstances of midwifery work in general practice; the paraphenalia and "perfection" of hospital procedures are not so adapted and never will be.

I have carried out no research on the vaginal flora, neither, as far as I can ascertain, has Dr. Jacobs, so that by his own implication he has no more right to comment on my remarks than I had to make them. Though diphtheritic, the Klebs-Löffler bacillus is not pathogenic to a carrier. If from the mass of conflicting evidence we conclude that pyogenic organisms are normally found in the healthy vagina, still my own experience justifies me in expressing my belief that pathogenic organisms are not present. We have, or course, some evidence that they are capable of becoming pathogenic if the continuity of the walls be broken. I, therefore, advocate the general adoption of procedures that will prevent the development of conditions at parturition which conduce to the laceration of cervix and perineum. This indeed is the main objective of my prophylactic practice.

Yours, etc.,

H. LEIGHTON KESTEVEN.

**Bullahdelah,**
**January 28, 1929.**

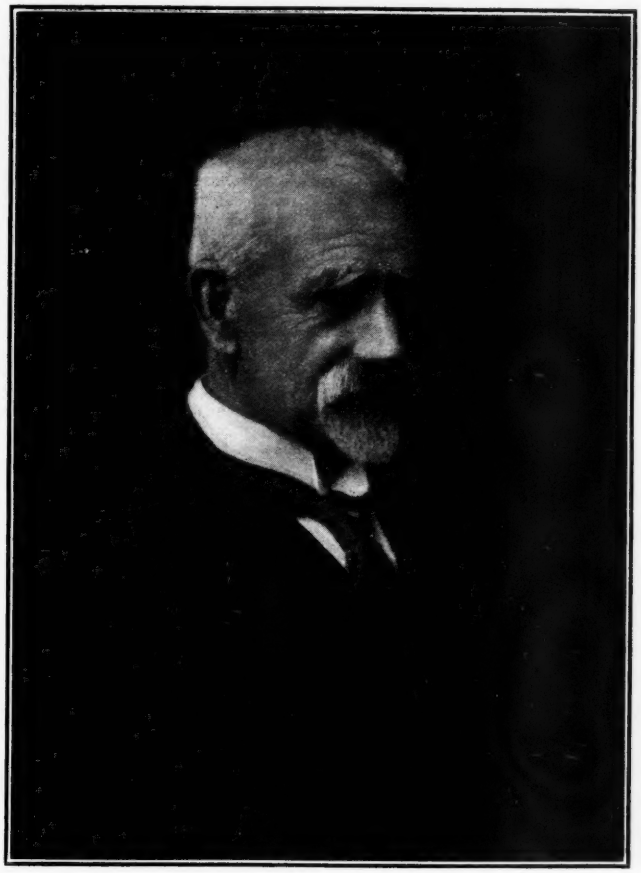
# Obituary.

## WILLIAM THORNBOROUGH HAYWARD.

WILLIAM THORNBOROUGH HAYWARD scarcely belonged to the present generation of medical practitioners. In these days of shaven faces and lounge suits we are apt to associate the courtly face and figure of Hayward with an age when diplomatic behaviour and fine breeding were associated with knee breeches and frilled shirts and when considerations for the susceptibilities and feelings of others were regarded as an essential attribute of a real gentleman. Hayward seems to have belonged to that age both in external appearance and in his courteous bearing. His passing hence at the ripe age of seventy-four is a sign that the modes and manners of one generation differ from those of the next and that the changes wrought by hurry and bustle may possibly be retrograde steps.

William Thornborough Hayward was born at Reading in England in 1854. His father lived on the banks of the Thames in a beautiful English town surrounded by the charming freshness of peaceful meadows and woodland. His boyhood was spent in the atmosphere of English rural life of the middle of last century. He went to school at Huntpierpoint in Sussex and in his early life he received the education and training characteristic of a well-bred English gentleman. At the age of fifteen years he entered the Royal Infirmary School of Medicine at Liverpool and passed the preliminary examination entitling him to become enrolled as a medical student. Two years later he distinguished himself by securing silver medals in medicine, surgery and pathology and an exhibition. Liverpool in those days was not a fashionable school of medicine, but the education given there was sound and of a practical nature. In accordance with the custom of those times he became apprenticed to a general practitioner prior to his qualification as a medical practitioner. His apprenticeship lasted for a year. He then was admitted to his final examination and in 1874 he obtained the diplomas of licentiate of the King's and Queen's College of Physicians of Ireland and licentiate in midwifery. He engaged himself at first as ship's surgeon on board mail steamers journeying to South America. After two voyages he returned to Liverpool and in 1875 he became house surgeon to Bickersteth, a surgeon at the Liverpool Royal Infirmary. At the same time he worked for and gained the diploma of member of the Royal College of Surgeons of England. In the following year he served as house surgeon at the Children's Hospital, Liverpool. These house appointments were valuable to him in giving him experience and confidence. In 1877 and 1878 he again went to sea as ship's surgeon, this time on the Australian route. His third voyage was made on an emigrant ship which reached Adelaide in October, 1878. The land of sunshine and endless promise attracted him and he stepped ashore to seek his fortune in a new land. At first he conducted a general practice at Riverton where he attained considerable success. He encountered small difficulty, for there was but little competition and his ability by this time was established. In 1882 he moved to Norwood and in building up his Adelaide practice he sought at an early date to devote his attention to medicine. He gained popularity among his colleagues and soon had their full confidence. The South Australian Branch of the British Medical Association had become recognized in the year 1880. Hayward had joined this great organization in England and while at Riverton he lent his support to a movement for the establishment of a branch in the Colony. The Branches in New South Wales, in Victoria and in South Australia were recognized simultaneously. Those in the other States have been established later. He was appointed to the Council of his Branch and in 1884 he was elected Vice-President. In the following year he was elected an honorary physician to the Adelaide Hospital, a position he retained, save for a period of five years, until 1914. In the same year he was selected to fill the President's chair in the South Australian Branch of the British Medical Association. In 1886 at the annual meeting of the Branch when Hayward vacated the chair, a proposal was made by the Council that a medical con-

gress should be established and that the first session should be held in Adelaide during the jubilee year of the reign of Queen Victoria. A committee was appointed to draw up a scheme and to make the necessary arrangements. William Thornborough Hayward was a leading member of this committee and his advice was freely given and as freely accepted. In December of the same year when the committee reported to the subscribers, Dr. (now Sir) Joseph Verco was appointed President of the first session of the Intercolonial Medical Congress and W. T. Hayward was appointed Honorary Treasurer. Dr. B. Poulton who had first suggested the institution of the congress, was the Honorary Secretary. The Honorary Treasurer apparently had a difficult task to make ends meet. In the records it is set forth that the total receipts including a grant from the Branch, a present from J. Davies Thomas and some sundry unusual contributions were £193 10s. 4d.! That was nearly forty-two years ago; few of the original committee in 1887 realized what they had succeeded in establishing.



In 1886 William Thornborough Hayward was appointed a lecturer in clinical medicine at the University of Adelaide. He was thirty-two years of age and had been in practice as a physician for but four years. His colleagues and the authorities recognized that this young man had won his way to the front rank by merit, hard work and a sound education and it is a tribute to his ability and erudition that he was able to hold these positions of responsibility and distinction until the outbreak of war when national claims overwhelmed everything else.

In 1888 he became lecturer in *materia medica* and therapeutics in addition to clinical medicine. In the following year he gained appointment as honorary medical officer to the Children's Hospital. During the years that followed, he worked conscientiously and well, enhancing his reputation as a physician at the hospitals, as a teacher at the university and as leader in the South Australian Branch of the British Medical Association. As a private practitioner his reputation increased and he certainly earned the respect and confidence of his very numerous patients.

In the year 1896 the medical profession in Adelaide found itself at variance with the Government in connexion with the arrangements at the Adelaide Hospital. The South Australian Branch of the British Medical Association determined to uphold its principles and adhere to the policy established in regard to the relations of the medical profession to the public hospitals. This was not the first time that the medical profession dared to alienate public opinion and to accept the financial loss resulting from its action in order to uphold a principle nor has it been the last. It will be remembered that the fight became a stern one when the Government engaged the services of men who were prepared to ignore the ethics of their profession and to accept positions on the hospital staff on conditions that had been declared by the vast majority of the members of the South Australian Branch to be unacceptable and derogatory to the honour and interests of the profession. It was realized that strong men were required to lead the profession during this critical period and with one accord his colleagues invited William Thornborough Hayward to accept the office of Honorary Secretary of the Branch. The struggle was an unequal one. Pitted against the handful of medical practitioners was a Government and the issue was transformed into a political one, since it was no concern of the politicians to deal with matters of ethics. Hayward proved himself a capable and safe guide during that difficult period and he heartened his colleagues to work patiently for a moral victory. Two years later when matters had improved, he relinquished his position to take over the duties of Honorary Treasurer of the Branch, a position he held for eleven years.

The fourth session of the Intercolonial Medical Congress of Australasia was held in Dunedin in February, 1896. Hayward acted as President of the Section of Medicine, a position of considerable importance. In 1899 he was appointed a member of the Council of the University of Adelaide. From this time he took an active part in the conduct of affairs of the University and for a quarter of a century he gave to the University very valuable services. He was a stern and unbending fighter and a formidable opponent and he held very strong views on many matters connected with the control of education and the functions of the University.

In the year 1905 the seventh session of the Australasian Medical Congress was held in Adelaide. William Thornborough Hayward was the Honorary Treasurer and as usual he took his duties seriously. Matters had developed greatly in the interval since the first session eighteen years before. The meeting was much larger and the organization was more complicated, although no fundamental alteration had been adopted. Hayward recognized that there would be a grave danger if the members at the final plenary session adopted important resolutions without adequate consideration and discussion. He therefore induced the executive committee to admit for discussion at the final meeting only those resolutions that had been adopted at meetings of the sections and sent to the final meeting with a recommendation for their adoption by the whole congress. Subsequent events have proved that he was wise in his endeavour to prevent the hurried adoption of resolutions at plenary sessions in circumstances that rendered it inevitable for the majority to be ignorant of the real significance of the resolution. This danger is so real that the Federal Committee in

drafting the constitution of the Australasian Medical Congress (British Medical Association) has deliberately omitted all provision for the discussion of matters of medical policy at final meetings.

The index of his influence on the affairs of the medical profession in South Australia is to be sought in the measure of the trust imposed by his colleagues on him. Twenty-five years after he had been selected to fill the chair of the Branch, he was again invited to occupy this position. In 1909 he became Vice-President of the South Australian Branch of the British Medical Association and in 1910 he was for the second time President. In the following year he was President of the Section of Medicine at the ninth session of the Australasian Medical Congress in Sydney. About this time the Federal Committee of the Branches of the British Medical Association in Australia was formed and he became its first chairman. When the history of the Federal Committee is written, the name of William Thornborough Hayward will occupy an important and prominent place. It is impossible in this place to refer in detail to the work that was accomplished while Hayward was chairman and Dr. G. H. Abbott was honorary secretary. The fact, however, needs emphasis, for it signifies that the trust imposed on him by his colleagues throughout Australia was sufficiently great to impel them to place him in the most coveted position in the world of medico-politics.

As early as 1880 Hayward had joined the military services as surgeon to the Riverton Company of the South Australian Volunteers. Later he had served as Surgeon-Major in the South Australian Militia. At the outbreak of war he offered his services to his country, despite his sixty years. He joined the Australian Imperial Forces and in 1915 he was appointed physician to the No. 9 Australian General Hospital with the rank of Major. A little later he was promoted to the rank of Lieutenant-Colonel and embarked for England as senior medical officer. At first he was placed in charge of the No. 10 Australian General Hospital and was then transferred to the headquarters staff. In November, 1915, he was appointed Officer in Command of the First Australian Auxiliary Hospital, Harefield. He continued in this position for nearly two years. In September, 1917, he returned to headquarters and was required to act as senior physician to the medical board. At this time he was mentioned in dispatches "for distinguished services rendered in connexion with the war." The date was July 27, 1917. Earlier in the same year he received the honour of appointment as Commander of the Order of Saint Michael and Saint George. Towards the end of the year he became senior physician at the No. 2 Australian General Hospital, Boulogne. He received his promotion to the rank of Colonel in 1918. After serving on the hospital transport *Balmoral Castle* for a time, he returned to Australia and his name was transferred to the lists of the Australian Military Forces. In 1921 he was on the staff of the Keswick Repatriation Hospital.

During the period of the war he was chosen by the South Australian and the Victorian Branches to represent them on the Council of the British Medical Association. It was while he was a member of the Council that he seized the opportunity of asking for steps to be taken to grant fuller autonomy to the Australian Branches. The negotiations then opened were not immediately successful, but at a later date Dr. R. H. Todd was able to bring convincing argument that there was neither legal nor constitutional impediment to prevent the Branches in Australia from becoming incorporated under the several *Companies Acts*. In many other ways William Thornborough Hayward furthered the interests of the Branches in Australia while he was a member of the Council of the British Medical Association. About this time the University of Aberdeen conferred on him the honorary degree of doctor of laws. At a subsequent date the University of Adelaide conferred on him the degree of LL.D. *ad eundem gradum*. He was appointed a Vice-President of the British Medical Association in 1914. This honour has been conferred on but two other medical practitioners in Australia and on but few in the United Kingdom and other parts of the Empire.

After his return to Australia he resumed his private practice, but did not again engage in hospital practice. In 1922 he was elected Dean of the Faculty of Medicine. He relinquished this position in 1923. Failing health and advancing years compelled him to restrict his activities from this time and for the remainder of his life he was in practical retirement. Of his achievements as a physician it is difficult to give an adequate conception in a few words. He held positive views; some have gained support, while others have been challenged. He belonged to the school of clinicians rather than of pathologists and in consequence the younger generations of students and medical graduates know but little of his opinions and teaching. He was a sound teacher and a good lecturer and his wide interest in his students endeared him to many successive "years" of students. He was keenly interested in the training of masseurs and also in the technical education of nurses. He had a thousand and one other interests. In 1922 Dr. W. N. Robertson asked the Federal Committee to institute a gold medal for distinguished service to the British Medical Association in Australia and in 1923 Sir George Syme was requested to make the first presentation. The medal was awarded to William Thornborough Hayward and to Robert Henry Todd. Sir George Syme delivered a fitting eulogy to both recipients on the occasion of the

first session of the Australasian Medical Congress (British Medical Association) in Melbourne. No further awards have been made up to the present time.

In his last long illness he was devotedly nursed by Mrs. Hayward to whom the sympathy of the whole medical profession is extended. Her husband was a man whose life and work have left an enduring influence on the status of the medical profession.

Sir Henry Newland writes:

My memories of Dr. Hayward, "W.T." as he was generally spoken of, date from my boyhood when he used to wave genially to me from his gig as I rode on horseback to school. Some years later I acted as medical clerk in his wards at the Adelaide Hospital. He was particularly interested in diseases of the heart and lungs and I recall what a pleasure it was to watch him percuss a chest, a procedure in which he took a special pride. Having a good ear for music, he was able with his long delicate fingers to elicit and detect very slight variations from the normal percussion note. Though he was a very sound physician and in his prime had a very large practice, it is chiefly for his medico-political work that Hayward will be remembered. It was largely his imagination and forcefulness that begat the Federal Committee and subsequently as its Chairman he fostered its activities. During the war he served on the Council of the British Medical Association in London and later on when I succeeded him, I was able to gauge the high regard the members of the Council had for him. Throughout the greater part of his professional life it was always a great source of regret to him that he did not possess a university degree. His gratification, therefore, was extreme when the University of Aberdeen conferred on him the honorary degree of LL.D. He found his chief recreation in cards and at one time belonged to a famous group of whist players in Adelaide. When bridge ousted that game, he played it with the same keen enjoyment. A sarcastic tongue, such as Hayward had, is apt to be a dangerous possession and it says much for his other more lovable qualities that it did not affect his friendships. For very many years he was the guide, philosopher and friend of the younger men who never sought his advice in vain. His infinite tact found a way out of many a difficult situation. He was indeed a very perfect gentleman and Time, the great eraser, will not easily efface the record of his achievements from the medical annals of Australia.

### GABRIEL WILLIAM STAHEL FARMER.

WE regret to have to announce the death of Dr. Gabriel William Stahel Farmer on February 1, 1929, at Maryborough, Queensland.

### FREDERICK THOMAS ALLEN.

IT is with regret that we learn of the death on February 2, 1929, of Dr. Frederick Thomas Allen, of South Kensington, near Sydney, New South Wales.

### CHARLES STANSER BOWKER.

WE regret to announce the death of Dr. Charles Stanser Bowker which occurred at Randwick, New South Wales, on February 7, 1929.

## Congress Notes.

### THE AUSTRALASIAN MEDICAL CONGRESS.

THE Executive Committee of the third session of the Australasian Medical Congress (British Medical Association), Sydney, 1929, announces that the Senate of the University of Sydney has granted the use of the Medical School and the University buildings for the period of the session.

#### Membership.

Those desiring to attend the session are requested to fill in the form of application for membership. This form was printed in THE MEDICAL JOURNAL OF AUSTRALIA of January 5, 1929.

#### The Sections.

The scientific work of the session will be divided into the following sections.

*Section of Anæsthetics*: President, Dr. Gilbert Brown, Gilberton, South Australia; Honorary Secretary, Dr. J. G. Hunter, Mascot, Sydney.

*Section of Dermatology*: President, Dr. R. R. Wettenhall, 85, Spring Street, Melbourne; Honorary Secretary, Dr. G. R. Hamilton, 147, Macquarie Street, Sydney.

*Section of Medical Literature and History*: President, Dr. A. A. Lendon, 66, Brougham Place, North Adelaide; Honorary Secretary, Dr. L. Cowlishaw, Gordon Road, Lindfield.

*Section of Medicine*: President, Dr. C. T. Ch. de Crespigny, 219, North Terrace, Adelaide; Honorary Secretary, Dr. Cotter Harvey, 235, Macquarie Street, Sydney.

*Section of Naval and Military and Air Medicine and Surgery*: President, Dr. R. M. Downes, 55, Collins Street, Melbourne; Honorary Secretary, Dr. R. W. W. Walsh, Everton Road, Strathfield.

*Section of Neurology and Psychiatry*: President, Dr. W. E. Jones, Spring Street, Melbourne; Joint Honorary Secretaries, Dr. R. A. Noble, 143, Macquarie Street, Sydney, Dr. J. A. L. Wallace, Callan Park, Rozelle.

*Section of Obstetrics and Gynæcology*: President, Dr. H. Jellett, Christchurch, New Zealand; Honorary Secretary, Dr. A. J. Gibson, 143, Macquarie Street, Sydney.

*Section of Ophthalmology*: President, Dr. D. D. Paton, St. George's Terrace, Perth; Honorary Secretary: Dr. W. M. C. MacDonald, 235, Macquarie Street, Sydney.

*Section of Orthopædic Surgery*: President, Dr. A. V. Meehan, Wickham Terrace, Brisbane; Honorary Secretary, Dr. J. Hoets, 143, Macquarie Street, Sydney.

*Section of Oto-Rhino-Laryngology*: President, Dr. R. Graham Brown, Preston House, Brisbane; Honorary Secretary, Dr. Garnet Halloran, 143, Macquarie Street, Sydney.

*Section of Pædiatrics*: President, Dr. H. D. Stephens, 41, Spring Street, Melbourne; Honorary Secretary, Dr. M. J. Plomley, 233, Macquarie Street, Sydney.

*Section of Pathology and Bacteriology*: President, Professor P. MacCallum, University of Melbourne; Honorary Secretary, Dr. F. S. Hansman, 143, Macquarie Street, Sydney.

*Section of Preventive Medicine and Tropical Hygiene*: President, Dr. R. C. Everitt Atkinson, Department of Public Health, Western Australia; Honorary Secretary, Dr. E. S. Morris, Department of Public Health, New South Wales.

*Section of Radiology and Medical Electricity*: President, Dr. H. M. Hewlett, 101, Collins Street, Melbourne; Honorary Secretary, Dr. H. M. Cutler, 139, Macquarie Street, Sydney.

*Section of Surgery*: President, ————; Honorary Secretary, Dr. T. Farranridge, 229, Macquarie Street, Sydney.

### DIPLOMA IN MEDICAL RADIOLOGY AND ELECTROLOGY.

THE University of Cambridge grants a diploma in medical radiology and electrology to medical graduates who have completed the prescribed courses and who have paid the prescribed fees. The candidate for examination in Part I must have attended a course of lectures and practical instruction in physics and electro-technics and an introductory course of clinical instruction in medical radiology and electrology for a period of three months. The candidate may present himself for Part II of the examination after he has completed a three months' course of clinical instruction and has attended clinical practice in the radiological department of a hospital approved for the purpose. He must further have studied medical radiology and electrology for a period of not less than six months in all and produce evidence to show that he has held his medical qualification for not less than one year.

A thesis in the form of a critical report on six cases either in radiology or in electrology or in both subjects must be submitted after the examinations have been passed. The candidate may be required to present himself for an oral examination. In exceptional circumstances no thesis will be required, but the candidate must have held a responsible post as radiologist for at least three months. The fees for the courses of lectures and practical work amount to forty-five guineas; those for each part of the examination are ten guineas and the fee on presenting the thesis is four guineas.

### LISTS OF MEMBERS.

IT has been decided to issue the lists of members of the several branches of the British Medical Association in Australia as a document apart from this journal. As the cost of setting and printing this list is considerable, the Directors of the Australasian Medical Publishing Company, Limited, have resolved to offer the copies for sale at one shilling each. Members desiring to receive a copy should send in their orders either to the Honorary Secretary of the Branch to which they belong or to the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales, together with a postal note for one shilling. The orders must be received not later than February 28, 1929.

## Medical Appointments.

Dr. Henry Raymond Hancock has been appointed Resident Medical Officer at the Mental Hospital, Parkside, South Australia.

* * *

Dr. Alec Letts Dawkins (B.M.A.) has been appointed Resident Medical Officer at the Babies' Hospital, Adelaide.

* * *

Dr. Edward Angas Johnson has been appointed Deputy Inspector-General of Hospitals, South Australia.

* * *

Dr. Robert Henry Pulleine (B.M.A.) and Dr. Albert Ray Southwood (B.M.A.) have been appointed Members of the Dental Board of South Australia.

* * *

Dr. Leslie Halse Rogers (B.M.A.) has been appointed Acting Honorary Ear, Nose and Throat Surgeon to the Coast Hospital, Sydney.

## Diary for the Month.

FEB. 19.—New South Wales Branch, B.M.A.: Organization and Science Committee.
FEB. 19.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
FEB. 22.—Queensland Branch, B.M.A.: Council.
FEB. 26.—New South Wales Branch, B.M.A.: Medical Politics Committee.
FEB. 26.—Section of Orthopædic Surgery, New South Wales Branch, B.M.A.
FEB. 27.—Victorian Branch, B.M.A.: Council.
FEB. 28.—South Australian Branch, B.M.A.: Branch.

## Medical Appointments Vacant, etc.

**For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xx.**

ADELAIDE HOSPITAL: Medical Registrar, Surgical Registrar.
ROYAL HOSPITAL FOR WOMEN, PADDINGTON: Resident Medical Officer.
WESTERN AUSTRALIAN STATE PUBLIC SERVICE: Medical Officer of Health.

## Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

| BRANCH. | APPOINTMENTS. |
|---|---|
| NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney. | Australian Natives' Association.<br>Ashfield and District Friendly Societies' Dispensary.<br>Balmain United Friendly Societies' Dispensary.<br>Friendly Society Lodges at Casino.<br>Leichhardt and Petersham Dispensary.<br>Manchester United Oddfellows' Medical Institute, Elizabeth Street, Sydney.<br>Marrickville United Friendly Societies' Dispensary.<br>North Sydney United Friendly Societies.<br>People's Prudential Benefit Society.<br>Phœnix Mutual Provident Society. |
| VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne. | All Institutes or Medical Dispensaries.<br>Australian Prudential Association Proprietary, Limited.<br>Mutual National Provident Club.<br>National Provident Association.<br>Hospital or other appointments outside Victoria. |
| QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane. | Members accepting appointments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing.<br>Brisbane United Friendly Society Institute.<br>Stannary Hills Hospital. |
| SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide. | All Contract Practice Appointments in South Australia.<br>Booleroo Centre Medical Club. |
| WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth. | All Contract Practice Appointments in Western Australia. |
| NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington. | Friendly Society Lodges, Wellington, New Zealand. |

Medical practitioners are requested not to apply for appointments to position at the Hobart General Hospital, Tasmania, without first having communicated with the Editor of THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales.

## Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, Sydney. (Telephones: MW 2651-2.)

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £1 for Australia and £2 5s. abroad *per annum* payable in advance.